“CLINICAL STUDY ON INCIDENCE, PATHOLOGICAL PATTERN AND MANAGEMENT OF GASTRIC CARCINOMA IN GOVERNMENT RAJAJI HOSPITAL, MADURAI”

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DECLARATION

I , DR.P.ANISH , solemnly declare that this dissertation titled **"CLINICAL STUDY ON INCIDENCE, PATHOLOGICAL PATTERN AND MANAGEMENT OF GASTRIC CARCINOMA IN GOVERNMENT RAJAJI HOSPITAL, MADURAI"** is a bonafide record of work done by me at the Department Of General Surgery, Government Rajaji Hospital , Madurai , under the guidance of **Dr.N.VIJAYAN, M.S,** Professor , Department of General Surgery, Madurai Medical college , Madurai.

This dissertation is submitted to The Tamil Nadu Dr. M.G.R Medical University, Chennai in partial fulfillment of the rules and regulations for the award of **M.S Degree General Surgery Branch- I**; examination to be held in **April 2015.**

Place: Madurai

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INTRODUCTION

INTRODUCTION

Early gastric cancer is defined as adenocarcinoma limited to the mucosa and submucosa of the stomach, regardless of lymph node status. The entity is common in the Orient, where gastric cancer is a common cause of cancer death, and where aggressive surveillance programs have therefore been established. Approximately 10% of patients with early gastric cancer will have lymph node metastases. There are several types and subtypes of early gastric cancer.

Approximately 70% of early gastric cancers are well differentiated, and 30% are poorly differentiated. The overall cure rate with adequate gastric resection and lymphadenectomy is 95%. In some Japanese centers, 50% of the gastric cancers treated are early gastric cancer. In the United States, less than 20% of resected gastric adenocarcinomas are early gastric cancer. Small intramucosal lesions can be treated with EMR.

Stomach cancer or **gastric cancer**, is when cancer develops from the lining of the stomach.[1] Early symptoms may include: heartburn, upper abdominal pain, nausea, and loss of appetite. Later symptoms may include: weight loss, yellow skin, vomiting, difficulty swallowing, and blood in the stool among others.[2] The cancer

may spread from the stomach to other parts of the body, particularly the liver, lungs, bones, lining of the abdomen and lymph nodes.[3]

The most common cause is infection by the bacteria *Helicobacter pylori*, which accounts for more than 60% of cases.[4][5] Certain type of *H. pylori* have greater risks than others. Other common causes include eating pickled vegetables, and smoking. About 10% of cases run in families and between 1% and 3% of cases are due to genetic syndromes inherited from a person's parents such as hereditary diffuse gastric cancer. Most cases of stomach cancers are gastric carcinomas. This type can be divided into a number of subtypes. Lymphomas and mesenchymal tumors may also develop within the stomach.

Most of the time, stomach cancer develops through a number of stages over a number of years.[5] Diagnosis is usually by biopsy done during endoscopy. This is than followed by medical imaging to determine if the disease has spread to other parts of the body.[2] At least Japan and South Korea, two countries that have high rates of disease, screen for stomach cancer.[5]

A Mediterranean diet lowers the risk of cancer as does the stopping of smoking. There is tentative evidence that treating *H. pylori* decreases the future risk.[5][6] If cancer is treated early many cases can be cured.[5] Treatments may include some combination of: surgery, chemotherapy, radiation therapy and targeted therapy.[2] If treated late palliative care may be advised.[5] Outcomes are often poor with a less than 10% 5-year survival rate globally. This is largely because most people with the condition present with advanced disease.[7] In the United States 5-year survival is 28%[8] while in South Korea it is over 65% partly due to screening efforts.[5]

PRECURSOR LESIONS OF CARCINOMA STOMACH

- CHRONIC ATROPHIC GASTRITIS
- ADENOMATOUS GASTRIC POLYPS
- INTESTINAL METAPLASIA

AIMS AND OBJECTIVES OF THE STUDY

1. To study the prevalence of carcinoma stomach as occurring in Government Rajaji Hospital, Madurai.

2. To study the clinical presentation including the anatomic site of occurrence and Histological type.

3. To study the association of risk factors

4. To study the surgical modalities of treatment

REVIEW OF LITERATURE

Stomach cancer is the fourth most common cancer diagnosed and the second most frequent cause of cancer death worldwide (1–2). Although stomach cancer rates are lower in the United States than worldwide generally, substantial numbers are affected. The American Cancer Society estimated that 21,500 people in the United States (13,190 men and 8,310 women) would be diagnosed with stomach cancer and 10,880 would die from the disease during 2008 (3). Incidence and mortality rates for stomach cancer in the United States have decreased steadily for many years (4–6).

Stomach cancer may be classified into intestinal and diffuse types based on histopathology, as initially described by Lauren (7). The two biological entities are different with regards to epidemiology, etiology, pathogenesis, and tumor behavior. The diffuse type occurs in relatively younger individuals and has a poorer prognosis compared with the intestinal type (8). Using the Surveillance, Epidemiology, and End Results (SEER) data from 1973 to 2000, Henson et al reported that rates for the intestinal type decreased about 50%, while rates for the diffuse or signet type rose more than 400% (9).

Stomach cancers also can be classified by subsite within the stomach: cardia, fundus, body, distal (antrum and pylorus), and lesser or greater curvature. Some studies have shown that there has been a striking increase in gastric cardia cancer in the United States since the 1970s, although the incidence of stomach cancer as a whole has decreased (10–11). Using the SEER-9 data from 1974–1976 to 1992–1994, Devesa et al (11) reported that the age-adjusted (1970 U.S. standard) incidence rates of gastric cardia cancer increased in both white males (from 2.1 to 3.3 per 100,000 person-years) and black males (from 1.0 to1.9 per 100,000 person-years). However, a more recent report using SEER-11 data for cases diagnosed during 1992–1998 found that gastric cardia rates did not significantly increase during that time period among any ethnic or gender group (12).

Expanding on previous studies and adding cancer cases diagnosed through 2005, we used SEER data to analyze stomach carcinoma incidence patterns by histologic type, anatomic site, race, gender, and age.

Gross Morphology and Histologic Subtypes

There are four gross forms of gastric cancer: polypoid, fungating, ulcerative, and scirrhous. In the first two, the bulk of the tumor mass is intraluminal. Polypoid tumors are not ulcerated; fungating tumors are elevated intraluminally, but also ulcerated. In the latter two gross subtypes, the bulk of the tumor mass is in the wall of the stomach.

Ulcerative tumors are self-descriptive; scirrhous tumors infiltrate the entire thickness of the stomach and cover a very large surface area. Scirrhous tumors (linitis plastica) have a particularly poor prognosis, and commonly involve the entire stomach. Although these latter lesions may be technically resectable with total gastrectomy, it is common for both the esophageal and duodenal margins of resection to show microscopic evidence of tumor infiltration. Death from recurrent disease within 6 months is common.

The location of the primary tumor in the stomach is important in planning the operation. Several decades ago, the large majority of gastric cancers were in the distal stomach. Recently, there has been a proximal migration of tumors, so that currently, the distribution is closer to 40% distal, 30% middle, and

30% proximal.

Histology

The most important prognostic indicators in gastric cancer are both histologic: lymph node involvement and depth of tumor invasion. Tumor grade (degree of differentiation: well, moderately, or poorly) is also important prognostically. There are several histologic classifications of gastric cancer. The World Health Organization recognizes several histologic types (Table 26-18). The Japanese classification is similar but more detailed. The commonly used Lauren classification separates gastric cancers into intestinal type (53%), diffuse type (33%), and unclassified (14%). The intestinal type is associated with chronic atrophic gastritis, severe intestinal metaplasia, and dysplasia, and tends to be less aggressive than the diffuse type. The diffuse type of gastric cancer is more likely to be poorly differentiated and is associated with younger patients and proximal tumors. The Ming classification also is useful and easy to remember, with only two types—expanding (67%) and infiltrative (33%).

Table 26-18 World Health Organization Histologic Typing of Gastric Cancer

Adenocarcinoma

Papillary adenocarcinoma

Tubular adenocarcinoma

Mucinous adenocarcinoma

Signet-ring cell carcinoma

Adenosquamous carcinoma

Squamous cell carcinoma

Small cell carcinoma

Undifferentiated carcinoma

Others

Source: Reproduced with permission from Ming S-C, Hirota T: Malignant epithelial tumors of the stomach, in Ming S-C, Goldman H (eds): *Pathology of the Gastrointestinal Tract,* 2nd ed. Baltimore: Williams & Wilkins, 1998.

Pathologic Staging

Ultimately, prognosis is related to pathologic stage. The most widespread system for staging of gastric cancer is the tumor-node-metastasis (TNM) staging system based on depth of tumor invasion, extent of lymph node metastases, and presence of distant metastases. This system was developed by the American Joint Committee on Cancer and the International Union Against Cancer, and has undergone several modifications since it was originally conceived

Stage grouping

Stage T N M

0 Tis N0 M0

IA T1 N0 M0

IB T1 N1 M0

T2 N0 M0

II T1 N2 M0

T2 N1 M0

T3 N0 M0

IIIA T2 N2 M0

T3 N1 M0

T4 N0 M0

IIIB T3 N2 M0

IV T4 N1–3 M0

T1–3 N3 M0

Any T Any N M1

CLASSIFICATIONS

1. LAURENS:
2. JAPANESE
3. BORMANNS
4. MINGS
5. WHO
6. SIEWERT FOR PROXIMAL GASTRIC CARCINOMA
7. MORPHOVOLUMETRIC
8. MORSON & DAWSON
9. KAJITANI

CLINICAL MANIFESTATIONS

Symptoms include

- abdominal pain,
- nausea and or or vomiting,
- weight loss,
- anorexia,
- jaundice,
- early satiety,
- dysphagia,
- malena and others.

Signs include

- anaemia,
- icterus,
- dehydration,
- ascites,
- visible gastric peristalsis &
- mass abdomen

LYMPH NODE GROUPS

GROUP 1 – PERIGASTRIC NODES

GROUP 2 – ALONG THE ROOT OF MAJOR VESSELS

GROUP 3 – AT THE ROOT OF SUPERIOR MESENTERIC ARTERY &

HEPATODUODENAL LIGAMENT

GROUP 4 –DISTANT LYMPH NODES

DIFFERENTIAL DIAGNOSIS

- ACID PEPTIC DISEASE
- PYLORIC STENOSIS WITH GASTRIC OUTLET OBSTRUCTION
- GASTRITIS
- PANCREATIC MASS – CARCINOMA
- TRANSVERSE COLON MASS – CARCINOMA
- ADVANCED FIXED STOMACH MASS MAY MIMIC RTROPERITONEAL OR NODAL MASS

LEATHER BOTTLE STOMACH

- ALSO CALLED LINITIS PLASTICA
- AN AGGRESSIVE DIFFUSE TYPE OF CARCINOMA STOMACH
- MOTHER OF PEARL APPEARANCE
- TYPE 4 GASTRIC CARCINOMA
- POORLY DIFFERENTIATED TYPE LACKING GLANDULAR FORMATION
- SPREADS SUBMUCOSALLY
- COMMON IN YOUNG, FEMALES, FAMILIAL, BLOOD GROUP A.
- SMALL SIZED STOMACH IN BARIUM MEAL STUDY
- TOTAL GASTRECTOMY WITH ESOPHAGO JEJUNAL ANASTAMOSIS.

BARIUM MEAL FINDINGS INN CARCINOMA STOMACH

1. IRREGULAR FILLING DEFECT
2. LOSS OF RUGOSITY
3. DELAYED EMPTYING
4. DILATATION OF STOMACH IN CARCINOMA PYLORUS
5. DECREASED STOMACH CAPACITY IN LINITIS PLASTICA
6. MARGIN OF THE LESION PROJECTS OUTWARDS FROM THE ULCER / LESION INTO THE GASTRIC LUMEN – CARMANNS MENISCUS SIGN.

DIAGNOSTIC EVALUATION

Distinguishing between peptic ulcer and gastric cancer on clinical grounds alone is usually impossible. Patients over the age of 45 years old who have newonset dyspepsia, as well as all patients with dyspepsia and alarm symptoms (weight loss, recurrent vomiting, dysphagia, evidence of bleeding, or anemia) or with a family history of gastric cancer should have *prompt upper endoscopy and biopsy* if a mucosal lesion is noted. Essentially, all patients in whom gastric cancer is part of the differential diagnosis should have endoscopy and biopsy. If suspicion for cancer is high and the biopsy is negative, the patient should be re-endoscoped and more aggressively biopsied. In some patients with gastric tumors, upper GI series can be helpful in planning treatment. Although a good *double-contrast barium upper GI examination* is sensitive for gastric tumors (up to 75% sensitive), in most centers, endoscopy has become the gold standard for the diagnosis of gastric malignancy.

Preoperative staging of gastric cancer is best accomplished with abdominal/pelvic CT scanning with IV and oral contrast. MRI is probably comparable. The best way to stage the tumor locally is via EUS, which gives fairly accurate (80%) information about the depth of tumor penetration into the gastric wall, and can usually show enlarged (>5 mm) perigastric and celiac lymph nodes. In some centers, if the tumor is transmural (T3) or involves lymph nodes

(enlarged nodes can usually be needled under ultrasound guidance), preoperative (neoadjuvant) chemotherapy is given. However, there are limitations to tumor staging with EUS. It largely is operator dependent and may underestimate lymph node involvement because normal-sized nodes (<5 mm) can harbor metastases. EUS is most accurate in distinguishing early gastric cancer (T1) from more advanced tumors.

PREOPERATIVE PREPARATION

- CORRECTION OF ANAEMIA, NUTRITION, FLUID & ELECTROLYTE.
- CARDIAC, RESPIRATORY & RENAL STATUS ASSESSMENT
- STOMACH WASH USING NORMAL SALINE
- PROPHYLACTIC ANTIBIOTIC AS ACHLORHYDRIA IN GASTRIC LUMEN ALLOWS COLONISATION OF STREPTOCOCCUS FECALIS, E.COLI, BACTEROIDES, STAPHYLOCOCCUS ALBUS.
- BLOOD / FFP MAY BE NEEDED PREOPERATIVELY AND FOR SURGERY.

Staging Laparoscopy and Peritoneal Cytology

To some extent, the usefulness of these modalities depends on the individual patient's situation as well as the treatment philosophy of the cancer team. The fundamental question is "will it make a difference to this patient's management?" Patients with gastric cancer who undergo R0 resection (i.e., no gross residual disease) and are found to have positive peritoneal cytology (no gross carcinomatosis) have a much worse prognosis than the cytology negative group (median survival 14.8 months vs. 98.5 months).96 It is controversial how much this information adds prognostically to that of pathologic staging (TNM). Whether this poor prognosis can be improved postresection with aggressive adjuvant treatment (systemic, or local intraperitoneal hyperthermic chemotherapy) is unknown. Unfortunately, it is also unclear how much these patients benefit from gastric resection. Currently peritoneal cytology information is unlikely to change the treatment of patients with gastric cancer, and most patients without detectable distant metastases will have (and should have) gastric resection regardless of the peritoneal cytology results. A quick laparoscopic examination can occasionally reveal small peritoneal implants or liver metastases that were not detected on preoperative imaging studies and, in some patients (e.g., high risk for surgery or impressive

carcinomatosis), this will change the operative plan and avoid a major but futile surgical procedure. Laparoscopy may be most useful in patients with proximal tumors or with adenopathy on spiral CT scan.97 An extensive laparoscopic staging procedure, although quite accurate, has not been widely adopted.

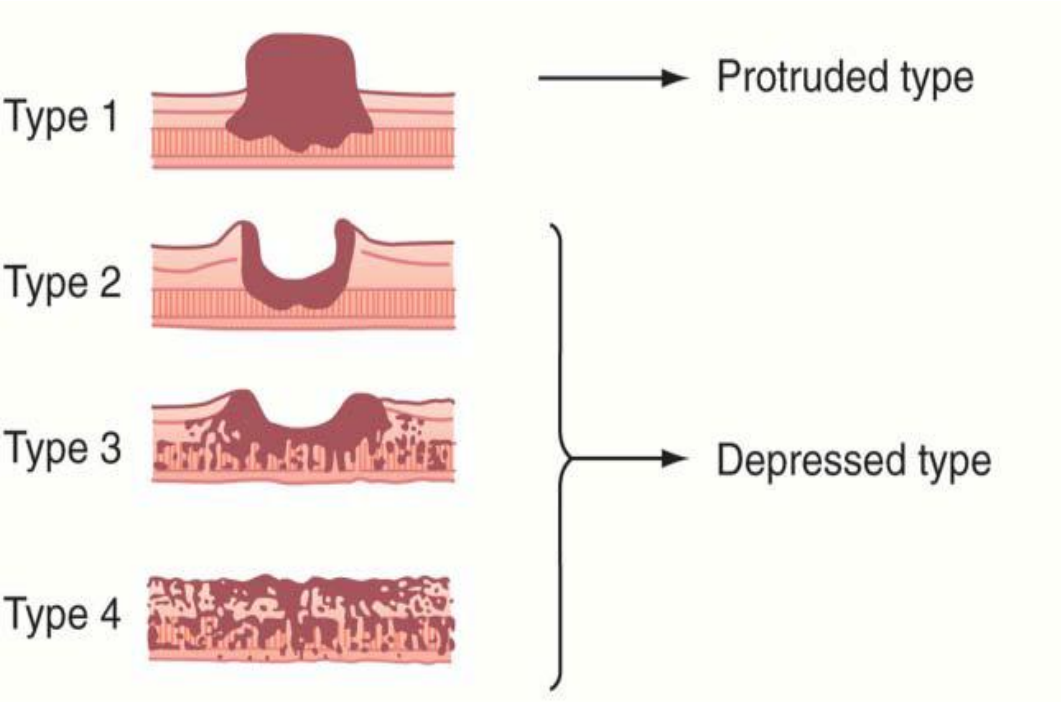
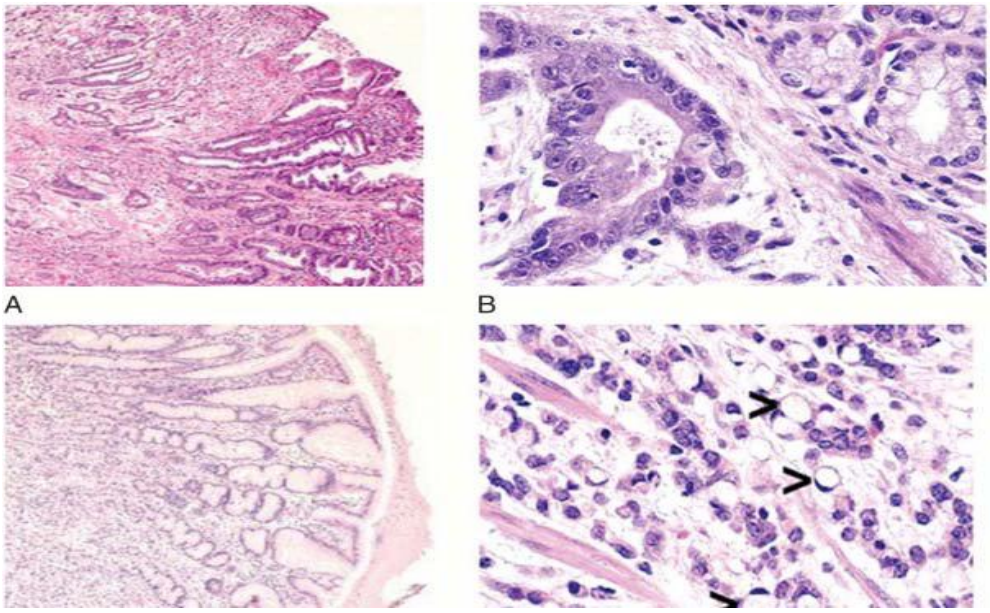


FIG: CARCINOMA STOMACH – BORMANN CLASSIFICATION



HISTOGRAPHIC PICTURES OF Adenocarcinoma.

Table 1- Lauren's Classification System

INTESTINAL	DIFFFUSE
Environmental	Familial
Gastric Atrophy, metaplasia	Blood group A
Men>Women	Women>men
Increasing incidence with age	Younger age group
Gland formation	Poorly differentiated, signet ring cells
Hematogenous spread	Transmural/lymphatic spread
Microsatellite instability, APC gene	Decrease E-cadherin mutations

Table 2 - Genetic Abnormalities in Gastric Cancer

ABNORMALITIES	GENE	APPROXIMATE FREQUENCY
Deletion/suppression	P53	60-70
	FHIT	60
	APC	50
	DCC	50
	E- cadherin	<5
Amplification/overexpression	COX	70
	HGF/SF	60
	VEGF	50
	c-met	45
	AIB -1	40
	Catenin	25
	k- sam	20
	Ras	10-15
	c-erb B2	5-7
Microsatellite instability		25-40
DNA aneuploidy		60-75

Fig.3 The role of Helicobacter Pylori

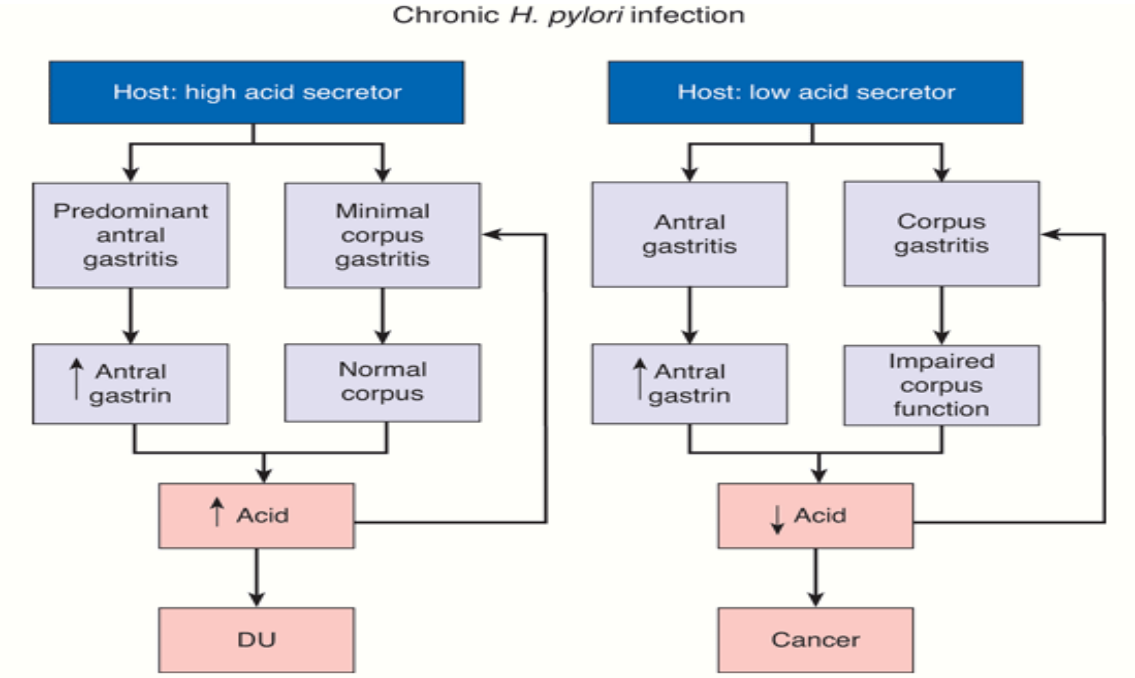


Fig .4 T Stage defined by depth of penetration into gastric wall

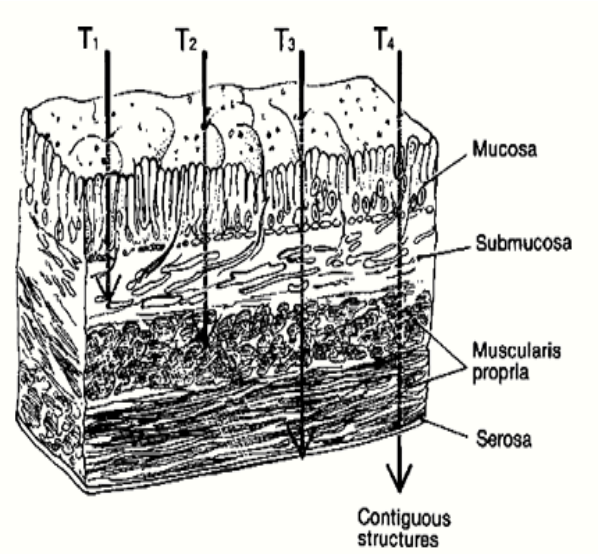


Fig 5 Lymph node station numbers as defined by Japanese Cancer Association

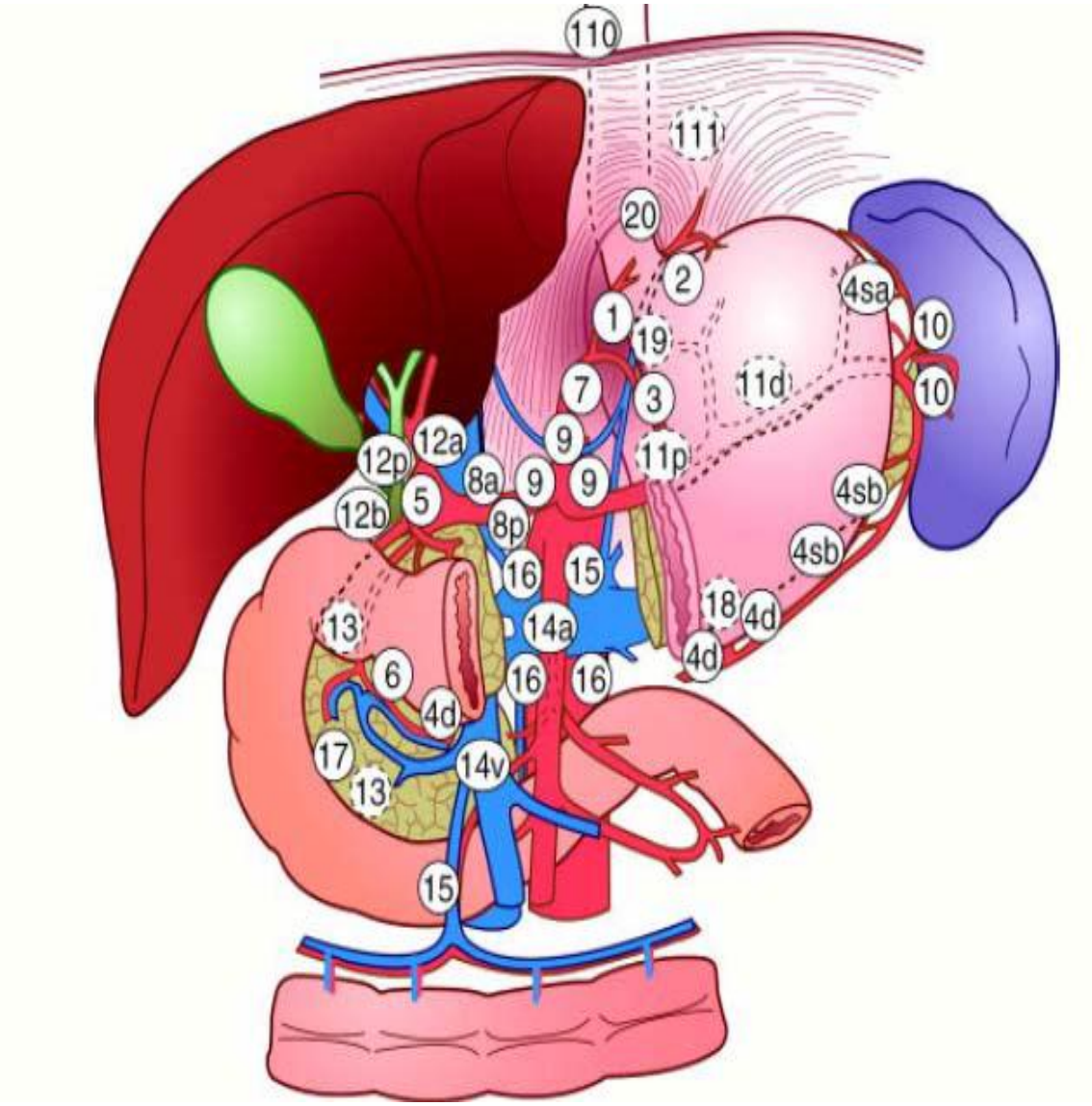


Table 6 - Grouping of Regional Lymph Nodes (Groups 1-3) by Location of Primary Tumor According to the Japanese Classification of Gastric Carcinoma

		LOCATION OF PRIAMRY TUMOUR IN THE STOMACH		
LYMPH NODE STATION(No	DESCRIPTION	UPPER THIRD	MIDDLE THIRD	LOWER THIRD
1	Right paracardial	1	1	2
2	Left paracardial	1	3	M
3	Lesser curvature	1	1	1

4sa	Short gastric	1	3	M
4sb	Left gastroepiploic	1	1	3
4d	Right gastroepiploic	2	1	1
5	Suprapyloric	3	1	1
6	Infrapyloric	3	1	1
7	Left gastric artery	2	2	2
8a	Anterior comm. hepatic	2	2	2
8p	Posterior comm. hepatic	3	3	3

9	Celiac artery	2	2	2
10	Splenic hilum	2	3	M
11p	Proximal splenic	2	2	2
11d	Distal splenic	2	3	M
12a	Left hepatoduodenal	3	2	2
12b,p	Posterior hepatoduodenal	3	3	3
13	Retropancreatic	M	3	3
14v	Superior mesenteric vein	M	3	2

14a	Superior mesenteric artery	M	M	M
15	Middle colic	M	M	M
16a1	Aortic hiatus	3	M	M
16a2,b1	Para-aortic, middle	M	3	3
16b2	Para-aortic, caudal	M	M	M

M, lymph nodes regarded as distant metastasis

BIRMINGHAM STAGING

1- CONFINED TO MUCOSA / MUSCULARIS PROPRIA

2- MUSCULARIS / SEROSAL INVOLVEMENT

3- NODAL SPREAD

4a- RESIDUAL DISEASE

4b- METASTATIC DISEASE

Spread of carcinoma of the stomach

No better example of the various modes by which carcinoma spreads can be given than the case of stomach cancer. It is important to note that this distant spread is unusual before the disease spreads locally and distant metastases are uncommon in the absence of lymph node metastases. The intestinal and diffuse types of gastric cancer spread differently. The diffuse type spreads via the submucosal and subserosal lymphatic plexus and it penetrates the gastric wall at an early stage.

Unlike malignancies such as breast cancer, nodal involvement does not imply systemic dissemination.

Blood-borne metastases

This occurs first to the liver and subsequently to other organs including lung and bone. This is uncommon in the absence of extensive nodal disease.

Trans peritoneal spread

This is a common mode of spread once the tumour has reached the serosa of the

stomach and indicates incurability. Tumours can manifest anywhere in the peritoneal cavity and commonly give rise to ascites. Advanced peritoneal disease may be palpated either abdominally or rectally as a tumour ‘shelf’.

The ovaries may sometimes may be the sole site of transcoelomic spread (Krukenberg’s tumours). Tumour may spread via the abdominal cavity to the umbilicus (Sister Joseph’s nodule). 25

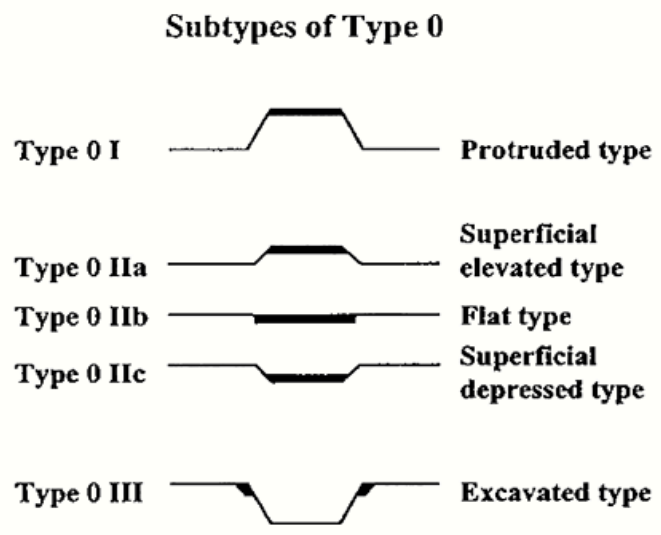
TREATMENT OF LOCALIZED DISEASE

STAGE I DISEASE (EARLY GASTRIC CANCER) - Classification and Risk for Nodal Metastases

The Japanese Research Society for Gastric Cancer has classified early gastric cancers (EGC) based on endoscopic criteria first established by the Japanese Endoscopy Society for the description of T1 tumors.

The current classification system is used for both in situ and invasive tumors and categorizes tumors based on endoscopic findings as follows:

Figure 6 . Japanese classification system for early gastric cancer. In the combined superficial types, the type occupying the largest area should be described first, followed by the next type (e.g., IIc + III). Type 0I and Type 0IIa are distinguished as follows: Type 0I: The lesion has a thickness of more than twice that of the normal mucosa. Type 0IIa: The lesion has a thickness up to twice that of the normal mucosa.



Considering the risk for lymph node metastasis is important when evaluating treatment options for patients with EGC. The frequency and anatomic distribution of nodal disease are related to the depth of tumor invasion. 10

Treatment options for patients with EGC include

- Endoscopic Mucosal Resection (EMR),
- Limited surgical resection,
- Gastrectomy.

Endoscopic Resection

It has been demonstrated initially at numerous East Asian centers that some patients with early gastric cancer can be adequately treated by an EMR. Small tumors (<3 cm) confined to the mucosa have an extremely low chance of lymph node metastasis (3%), which approaches the operative mortality rate for gastrectomy. If the resected specimen demonstrates no ulceration, no penetration of the muscularis mucosae, no lymphatic invasion, and size <3 cm, then the risk of lymph node metastases is less than 1%. Thus, some patients with early gastric cancer might be better treated with the endoscopic technique. Currently, this should be limited to patients with tumors <2 cm in size that are node negative and confined to the mucosa on EUS, in the absence of other gastric lesions. The addition of laparoscopic lymph node sampling may be considered in selected patients.

Limited Surgical Resection

Given the low rate of nodal involvement for patients with EGC, limited resection may be a reasonable alternative to gastrectomy for some patients with early EGC. There are no well-accepted pretreatment criteria for selection of patients for limited resection. Based on the existing pathology data, patients with small (less than 3 cm) intramucosal tumors and those with nonulcerated intramucosal tumors of any size may be candidates for EMR or limited resection. Surgical options for these patients may include gastrectomy with local excision.

Gastrectomy

Gastrectomy with lymph node dissection should be considered for patients with EGC who cannot be treated with EMR or limited surgical resection and/or patients who have intramucosal tumors with poor histologic differentiation or size greater than 3 cm or who have tumor penetration into the submucosa or beyond.

Gastrectomy with lymph node dissection allows for adequate pathologic staging and local therapy for these higher-risk patients. There is no consensus on the extent

of lymphadenectomy that should be performed as part of gastrectomy for EGC. Dissection of level I lymph nodes is a reasonable minimum standard at this time.

STAGE II AND STAGE III DISEASE

TREATMENT

Surgical resection is the only curative treatment for gastric cancer98,99 and most patients with clinically resectable locoregional disease should have gastric resection. Obvious exceptions include patients who cannot tolerate an abdominal operation, and patients with overwhelming metastatic disease.

The goal of curative surgical treatment is resection of all tumor (i.e., R0 resection). Thus, all margins (proximal, distal, and radial) should be negative and an adequate lymphadenectomy performed. Generally, the surgeon strives for a grossly negative margin of at least 5 cm. Some gastric tumors, particularly the diffuse variety, are quite infiltrative and tumor cells can extend well beyond the tumor mass; thus, gross margins beyond 5 cm may be desirable.

Frozen section confirmation of negative margins is important when performing operation for cure, but it is less important in patients with nodal metastases

beyond the N1 nodal basin. It should be strongly emphasized that many patients with positive lymph nodes are cured by adequate surgery. It should also be stressed that often lymph nodes that appear to be grossly involved with tumor turn out to be benign or reactive on pathologic examination. More than 15 resected lymph nodes are required for adequate staging.100 Therapeutic nihilism should be avoided and, in the low-risk patient, an aggressive attempt to resect all tumor should be made. The primary tumor may be resected en bloc with adjacent involved organs (e.g., distal pancreas, transverse colon, or spleen) during the course of curative gastrectomy. Palliative gastrectomy may be indicated in some patients with obviously incurable disease, but most patients presenting with stage IV gastric cancer can be managed without major operation.99,101

Extent of Surgery

Unless required for R0 resection, total gastrectomy confers no additional survival benefit and may have adverse nutritional or quality-of-life consequences, and higher perioperative morbidity and mortality.98,99 Subtotal gastric resection typically entails ligation of the left and right gastric and gastroepiploic arteries at the origin, as well as the en bloc removal of the distal 75% of the

stomach, including the pylorus and 2 cm of duodenum, the greater and lesser omentum, and all associated lymphatic tissue (Fig. 26-56). Reconstruction is usually by Billroth II gastrojejunostomy, but if a small gastric remnant is left (<20%), a Roux-en-Y reconstruction is considered. The operative mortality is around 2 to 5%. Radical subtotal gastrectomy is generally deemed to be an adequate cancer operation in most Western countries, provided that the contingencies stated result in tumor-free margins, >15 lymph nodes, and the resection of all gross tumor. In the absence of involvement by direct extension, the spleen and pancreatic tail are not removed.

Extent of Lymphadenectomy

The dialogue surrounding lymphadenectomy involves at least two important issues: (1) staging removal and histopathologic analysis of an adequate number of lymph nodes, and (2) therapy determining if some forms of lymphadenectomy are therapeutic for patients with gastric cancer. The current AJCC staging system (sixth edition) requires analysis of 16 or more lymph nodes to assign a pathologic N stage.

Adjuvant Therapy

The term adjuvant therapy is best used to describe additional treatment in an attempt to increase cure rates in patients who have already undergone a potentially curative resection. For gastric cancer, an R0 surgical procedure, in which all gross disease has been removed, the margins of resection are microscopically negative, and no distant metastases were found, is required before adjuvant therapy is considered. Resections that leave microscopic or gross residual disease are not adjuvant treatment, but rather therapy of known residual cancer.

The term perioperative chemotherapy (or neoadjuvant chemotherapy) involves the use of systemic treatment before definitive, potentially curative surgery.

Adjuvant Postoperative Systemic Therapy

Adjuvant postoperative systemic chemotherapy has not shown a significant advantage over surgery alone.

Postoperative Adjuvant Intraperitoneal Chemotherapy

Peritoneal recurrence is a common component of the failure pattern for patients with gastric cancer.

For gastric cancer, an increasing number of reports have been published in which immediate postoperative intraperitoneal therapy was given after potentially curative resection. However, no definitive conclusions can yet be drawn regarding the effectiveness of intraperitoneal postoperative chemotherapy in this setting.

Immunochemotherapy

Adjuvant immunostimulants given in association with cytotoxic chemotherapy has been studied, however larger studies that are adequately powered would be necessary to definitively evaluate this approach.

Perioperative (Neoadjuvant) Chemotherapy

Perioperative (pre- and postoperative) chemotherapy, also known as neoadjuvant chemotherapy, is an attractive concept in gastric cancer because many patients, particularly Western patients, have locally advanced tumors at diagnosis (T3 or T4, or obvious lymph node involvement).

Such patients are not only at substantial risk for distant metastasis, but local extent of the tumor may make an R0 resection difficult.

There are two goals to perioperative treatment: reduce the stage of the primary tumor to increase the likelihood that a R0 resection can be performed, and begin at an early time to treat micrometastatic disease.

The right gastric artery is ligated. At this point, the duodenum is divideddistal to the pylorus. The stomach and omentum are then reflected cephalad. The gastro hepatic ligament is divided close to the liver up to the gastro esophageal junction. Dissection is then continued on the hepatic artery toward the celiac axis.For tumors of the mid- and proximal stomach, dissection of the lymph nodes along the splenic artery and splenic hilum is important.

This technique is not indicated for antral tumors, given the low rate of splenic hilar nodal metastases seen with these tumors. The stomach is then divided 5 cm proximal to the tumor, which dictates the extent of gastric resection.

Despite the fact that the entire blood supply of the stomach has been interrupted, a cuff of proximal stomach invariably shows good vascularization from the feeding distal esophageal arcade.

TREATMENT OF ADVANCED DISEASE (STAGE IV)

Single-Agent Chemotherapy

For most drugs, a variety of doses and schedules have been studied. In the absence of comparative trials using the same agent with different doses and schedules, superiority of one regimen over the other cannot be assessed.

Combination Chemotherapy

Like other malignancies, multidrug regimens using agents that have a single-agent activity have been extensively studied in gastric cancer.

One of the most widely used combination chemotherapy regimens in upper gastrointestinal tract malignancies, including gastric cancer, is the two-drug combination of cisplatin and fluorouracil.

Other combination chemotherapy regimens include methotrexate, fluorouracil, and doxorubicin (FAMTX); etoposide, leucovorin, and fluorouracil combination; fluorouracil plus irinotecan; docetaxel added cisplatin fluorouracil; epirubicin-cisplatin-fluorouracil; irinotecan-fluorouracil-leucovorin ; Cisplatin Plus Irinotecan; Fluorouracil-Leucovorin-Oxaliplatin (FOLFOX)

Targeted Therapy

Bevacizumab

As with other solid tumors, including colorectal cancer, breast and lung cancers, therapeutic agents with a specific tumor target are now entering study in gastric and gastroesophageal junction tumors. One of the first compounds studied was bevacizumab, a humanized monoclonal antibody that binds the vascular endothelial growth factor ligand. Bevacizumab can safely be given with cytotoxic chemotherapy, including in patients in whom the primary tumor was still in place.

Epidermal Grow th Factor Receptor Tyrosine Kinase Inhibitors

Both erlotinib and gefitinib have been studied in gastroesophageal junction and gastric cancers. The bulk of the data is currently available only an abstract form. Cetuximab, an antibody to the epidermal growth factor receptor, is undergoing study as a single agent and also in combination with systemic cytotoxic chemotherapy.

Long-Term Side Effects of Therapy

Relatively little has been written in the oncology literature on the local effects of therapy for gastric cancer and its long-term implications. The presence of dumping syndrome is well known, with its resulting diarrhea and cramping, but vasomotor effects such as palpitations and diarrhea also occur either very shortly after a meal or 1 to 3 hours later. These symptoms probably result from rapid transit of food from the stomach into the small bowel with the release of various gastrointestinal hormones. These symptoms can be managed by adjusting the volume of oral intake and other dietary manipulations. There is also a reactive hypoglycemia that can result from the rapid insulin release after a meal with little gastric reservoir.In addition to dumping, there are a number of malabsorption issues that can be important. B malabsorption is well known, and many patients 12 are placed on monthly supplements, even if it is not clearly needed.

After partial gastrectomy one can often monitor B levels and replace 12 when needed. Iron and calcium absorption is improved by the gastric acid that is eliminated or decreased by surgery or radiation therapy, and bypassing the duodenum also decreases absorption. Low iron is discovered when patients develop an iron deficient anemia, but calcium malabsorption may not be found for many

years when the patient develops osteopenia. Patients should be followed with dual energy x-ray absorptiometry scans and/or placed on calcium supplementation (calcium citrate is better absorbed than calcium carbonate).

METHODOLOGY

The tissue for diagnosis was obtained by endoscopy or following surgical resection.

Source of Data

Patients presenting to Govt. Rajaji Hospital, Madurai during the study period and those found eligible were included in the study.

Sample size: Minimum of 50 cases meeting criteria of the present study

Inclusion Criteria

- Only patients with histological proven carcinoma stomach were included

Exclusion criteria

- Patients with tumour recurrence

COLLABORATING DEPARTMENTS:

Departments of General surgery,

Department of Surgical Gastroenterology,

Department of surgical oncology,

STUDY PERIOD: 12 months

INVESTIGATIONS

Routine Blood investigations like Haemoglobin%, Total Count, Differential Count, Bleeding Time, Clotting time.

Renal parameters like Blood Urea and Serum Creatinine

Liver Function Tests

Serum Electrolytes

Chest X-Ray

Blood Grouping

Special investigations like Upper Gastointestinal Endoscopy,

USG Abdomen

CT Scan Abdomen

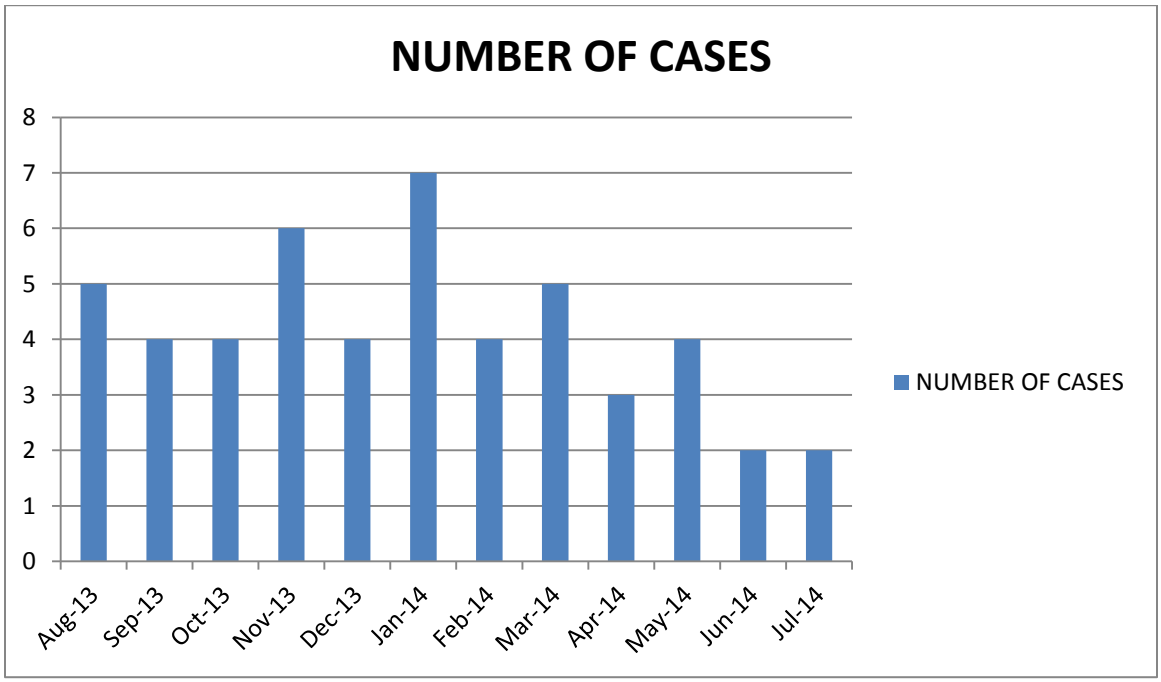
The cases were studied with importance given to clinical history regarding nature of presentation including diet history. The study of association of risk factors was also undertaken. Thorough clinical examination, Ultrasonography, endoscopy, CT in few cases and histopathologic diagnosis formed the basis of the study. The anatomic site of occurrence, the macroscopic type and the histopathologic type were studied in each case. An earnest attempt was made to study all the cases in detail with serial follow-up, the latter being incomplete due to non-responsive patients.

RESULTS

RESULTS

ANNUAL PREVALENCE: Graph-1

Gastric carcinoma is a common cancer with almost evenly distributed annual prevalence.

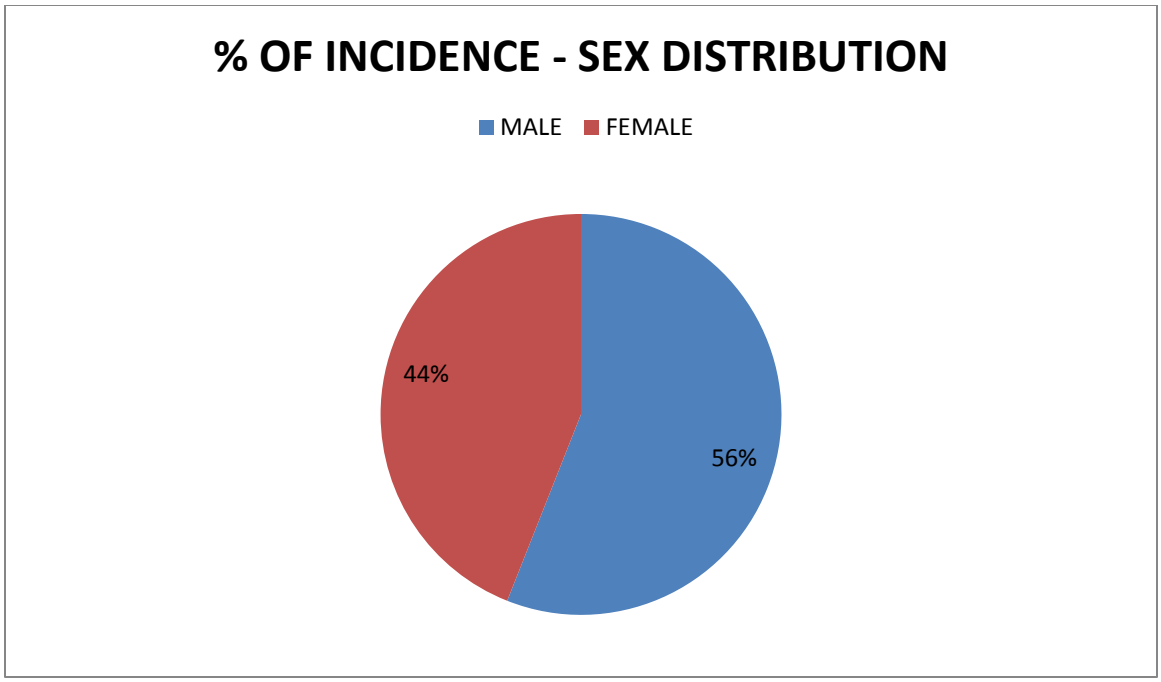


SEX PREVALENCE: Table 7; Graph 2

Table-7. Sex distribution among carcinoma stomach patients

Gastric cancer is more common in males with 56% of the cases being males in this study.

SEX	PRESENT STUDY	
	CASES	%
MALE	28	56
FEMALE	22	44

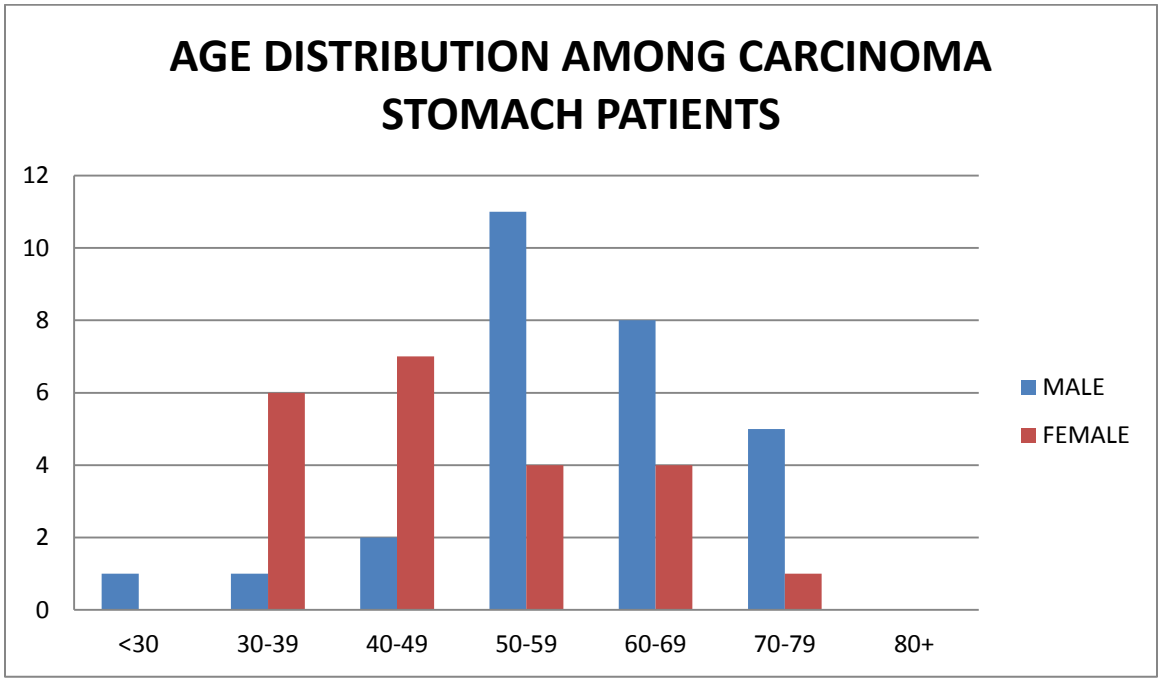


AGE PREVALENCE: Table 8; Graph 3

Table-8. Age distribution among carcinoma stomach patients

AGE	PRESENT STUDY			
	TOTAL	%	M	F
<30	1	2	1	0
30-39	7	14	1	6

40-49	9	18	2	7
50-59	15	30	11	4
60-69	12	24	8	4
70-79	6	12	5	1
80+	0	0	0	0

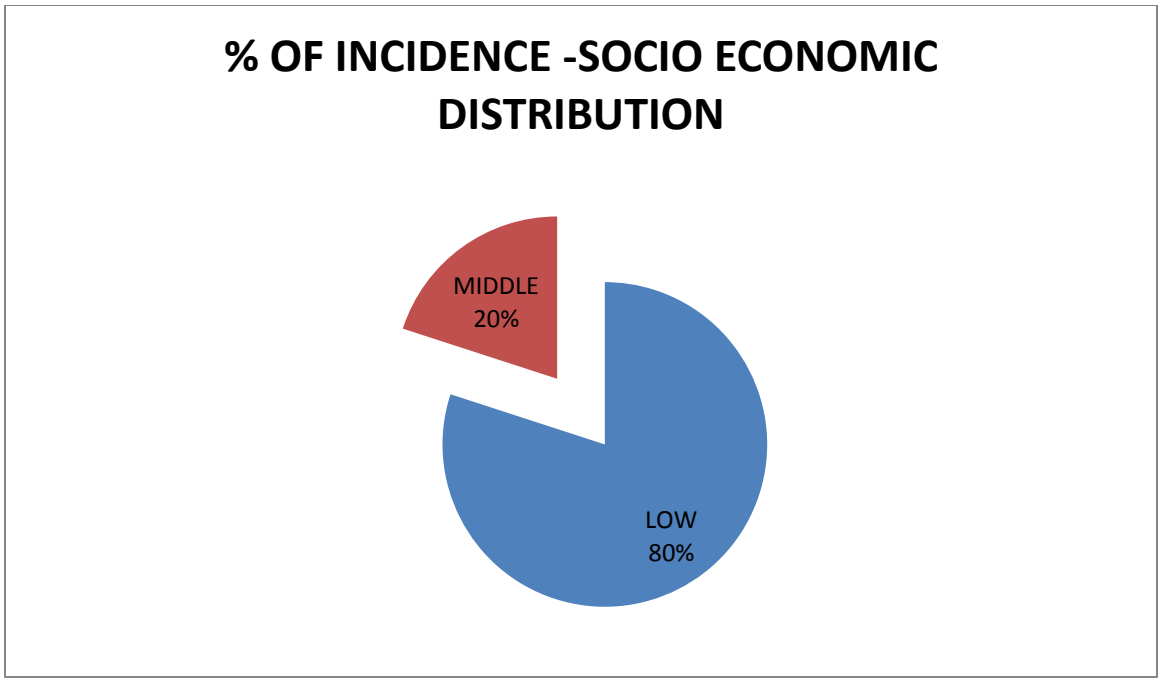


SOCIOECONOMIC STATUS: Table 9; Graph 4

The prevalence among the high socioeconomic group could not be studied as none of the patients belonged to this strata.

Table 9- Income group among carcinoma stomach patients

INCOME GROUP	PRESENT STUDY	
	CASES	%
LOW	40	80
MIDDLE	10	20
HIGH	0	0



RISK FACTORS: Table 10; Graph 5.0, 5.1

There are strong suggestions of the influence of environmental factors on gastric cancer. The most common risk factors associated were spicy food and mixed diet followed by tobacco and alcohol use.

In this study 45 (90%) patients consumed mixed diet and the rest were vegetarians. The non vegetarians took meat/fish approx. thrice every week. All patients in the study group frequently and regularly consumed green leafy vegetables.

Fruit consumption was frequent only in 40 (80%) of the cases.

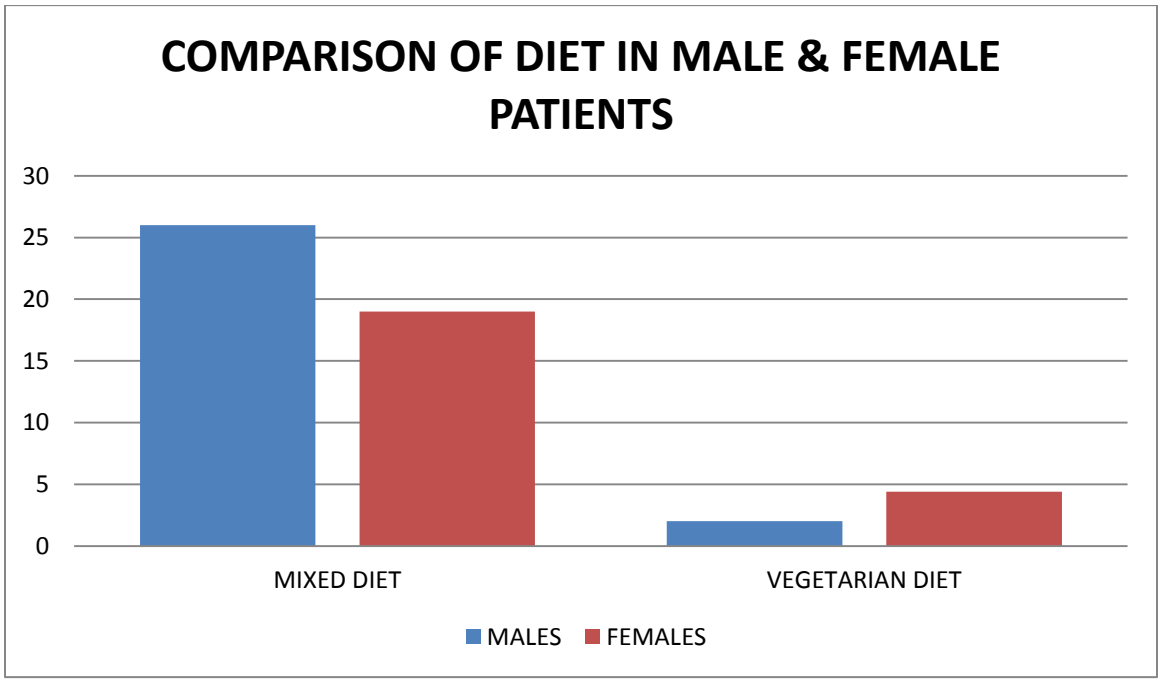
Smoked foods though a common risk factor in many countries was consumed only by 15 (30%) of the patients and even in theses patients the intake was not frequent. Majority of the patients, 40 (80%) reported to the use of high spicy diet in everyday food.

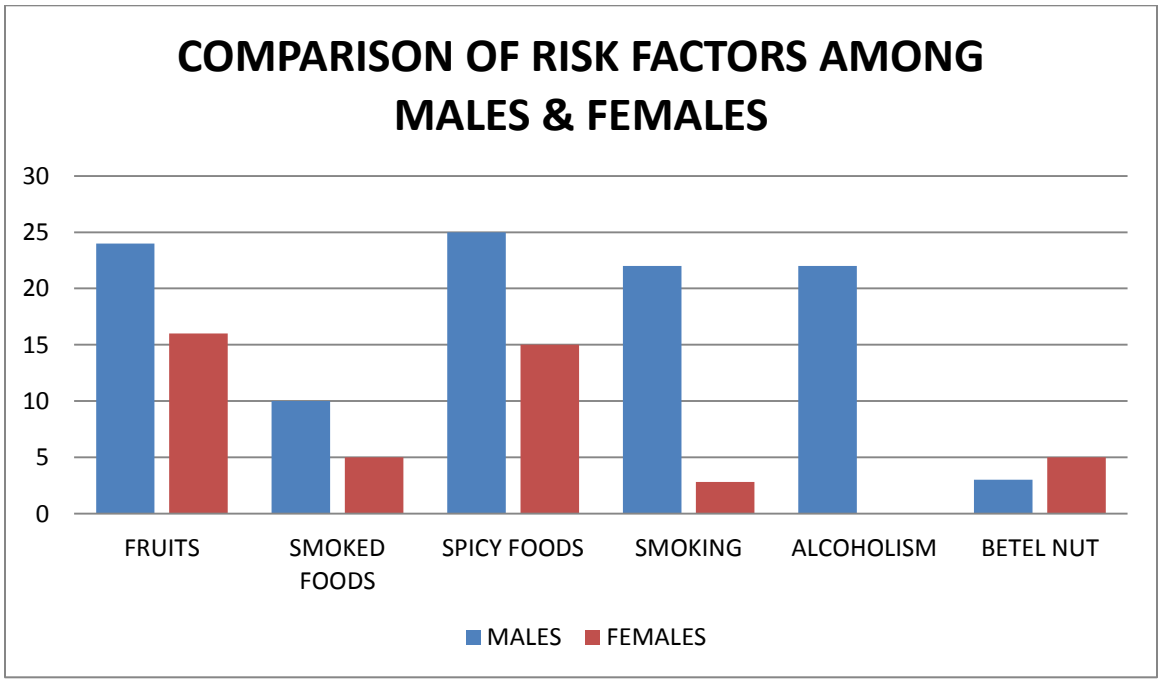
Tobacco smoking in the form of cigarette and beedi smoking was seen in 22 (44%) patients, all being males. Five females and three males reported to frequent use of betelnut which has been shown to a risk factor in the development of gastric cancer.

Alcohol consumption was seen in 22 (44%) of the patients, all males, who consumed it regularly and for a period of more than ten years.

Table 10 - Comparision of risk factors between males and females

RISK FACTORS	TOTAL		MALES		FEMALES	
	CASES	%	CASES	%	CASES	%
MIXED DIET	45	90	26	92.8	19	86.3
VEGETARIAN DIET	5	10	2	7.2	3	13.7
GREEN LEAFY	40	80	25	89.2	15	68.1
FRUITS	40	80	24	85.7	16	72.7
HIGH SALT INTAKE	32	64	20	71.4	12	54.5
SMOKED FOODS	15	30	10	35.7	5	22.7
SPICY	40	80	25	89.2	15	68.1
SMOKING	22	44	22	78.5	0	0
ALCOHOLISM	22	44	22	78.5	0	0
BETEL NUTS	8	16	3	10.7	5	22.7


COMPARISON OF DIET IN MALE & FEMALE PATIENTS
30
25
20
15
10
5
0
MIXED DIET
VEGETARIAN DIET
MALES
FEMALES

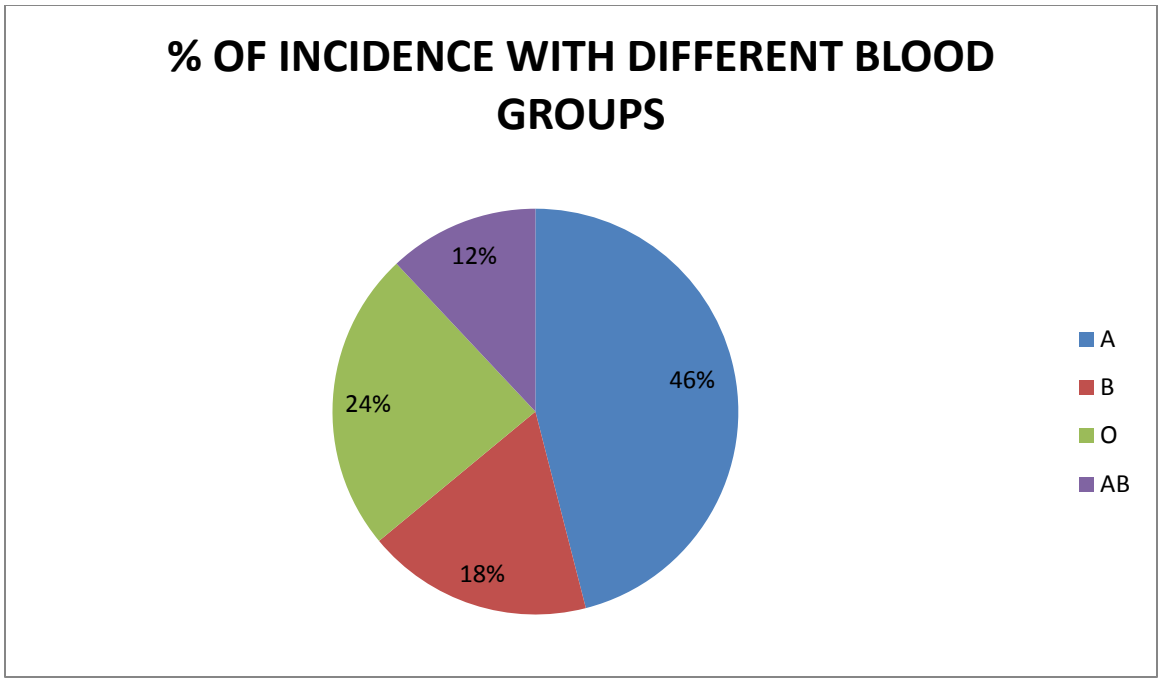

COMPARISON OF RISK FACTORS AMONG MALES & FEMALES
30
25
20
15
10
5
0
FRUITS
SMOKED FOODS
SPICY FOODS
SMOKING
ALCOHOLISM
BETEL NUT
MALES
FEMALES

BLOOD GROUP: Table 11; Graph 6

Blood group A showed the highest association with gastric cancer patients with 23 (46%) cases followed by blood group O and B.

Table 11 - Blood group distribution in carcinoma stomach patients

STUDY	BLOOD GROUP			
	A	B	O	AB
PRESENT STUDY	23 (46%)	9 (18%)	12 (24%)	6 (12%)



SYMPTOMS: Table 12, Graph 7

Anorexia was the most common symptom in patients and was reported in 42 (84%) of the patients. The next most common symptom was nausea and vomiting reflecting the high prevalence of distal tumours. 36(72%) reported weight >10% of body weight.

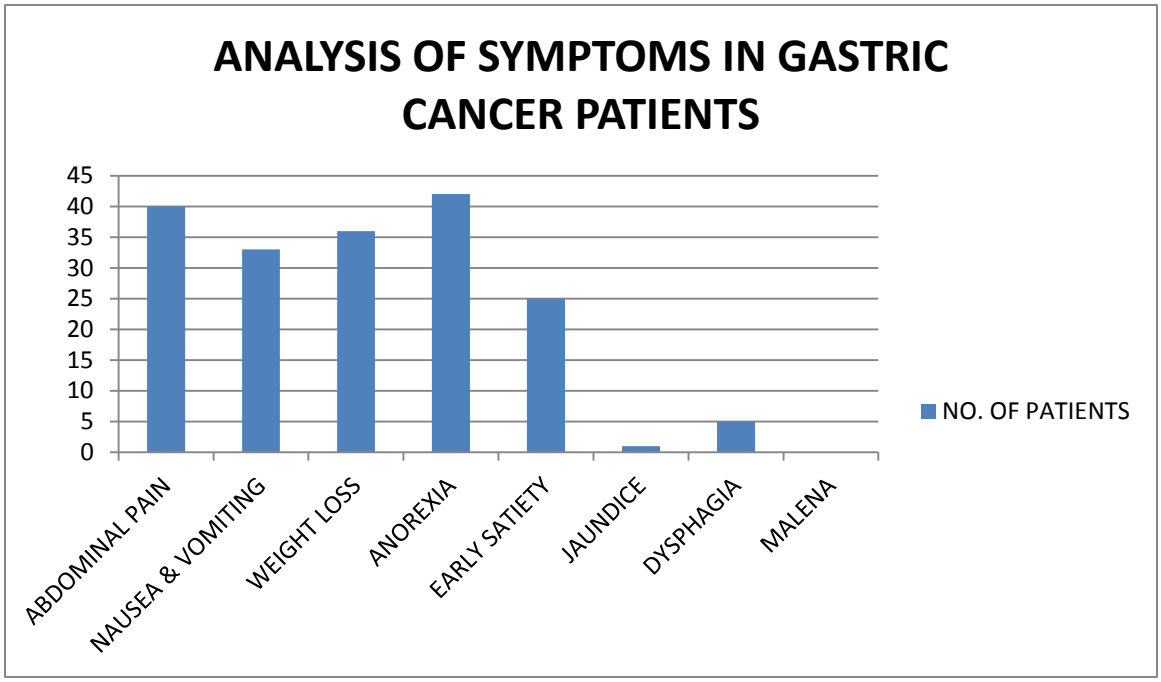
Proximal tumours involving the gastooesophageal junction had dysphagia as the predominant symptom. Only one patient in this study presented with jaundice and none of the patients had supraclavicular lymphadenopathy at presentation. One patient presented with features of peritonitis and was found to have a growth in the body of the stomach which had perforated.

Early satiety was reported in 25 (50%) of the patients which is characteristic of tumours involving the stomach wall diffusely.

Symptom analysis among the two sexes revealed that nausea, vomiting and weight loss were the most common symptoms followed by pain abdomen in females. The most common symptoms in males were anorexia, abdominal pain followed by nausea and vomiting.

Table 12-Symptom analysis in patients of carcinoma stomach

SYMPTOMS	PRESENT STUDY		MALES (28)		FEMALES (22)	
	CASES	%	CASES	%	CASES	%
ABDOMINAL PAIN	40	80	23	82.1	17	77.2
NAUSEA & VOMITING	33	66	18	64.2	15	68.1
WEIGHT LOSS	36	72	20	71.4	16	72.7
ANOREXIA	42	84	22	78.5	20	90.9
EARLY SATIETY	25	50	15	53.5	10	45.4
JAUNDICE	1	2	1	3.5	0	0
DYSPHAGIA	5	10	2	7.1	3	10.7
MALENA	10	20	5	17.8	5	17.8



SIGNS: Table 13, Graph 8

Overall, anemia was the most common sign in 35 (70%) of the cases followed by dehydration and ascites.

Visible gastric peristalsis the characteristic sign of gastric cancer was seen only in 7 (14%) of the cases. Gastric cancer presented as mass abdomen in 17 (34%) cases.

In females, anemia and ascites were the most common symptoms. None of the females in this study had visible gastric peristalsis which was seen in 7 (25%) of

males.

Presentation with mass abdomen was commoner in females 10 (45.4%) than in males 7 (25%).

Ascites at presentation suggesting the advanced stage of the disease was more common in females compared to males.

Table 13 - Analysis of signs in gastric cancer patients

SIGNS	TOTAL CASES	%	MALES		FEMALES	
			CASES	%	CASES	%
ANAEMIA	35	70	21	75	14	63.6
ICTERUS	1	2	1	3.5	0	0
DEHYDRATION	30	60	18	64.2	12	54.5
ASCITIS	28	56	17	60.7	11	50
VGP	7	14	7	25	0	0
MASS ABDOMEN	17	34	7	25	10	45.4

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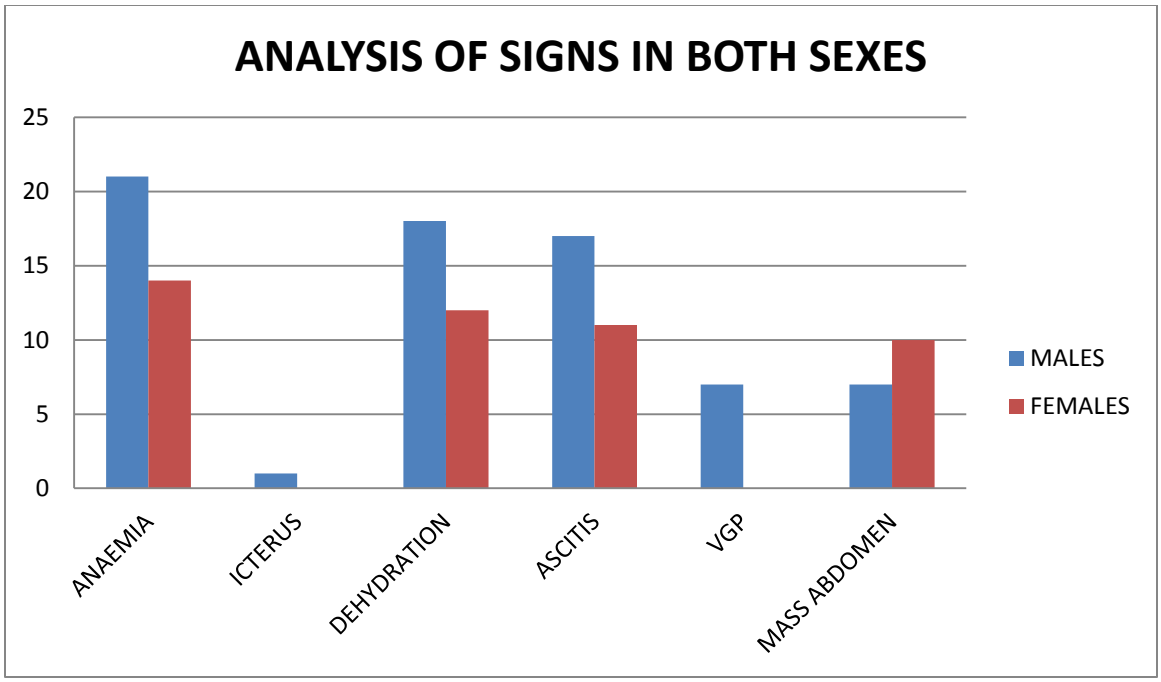
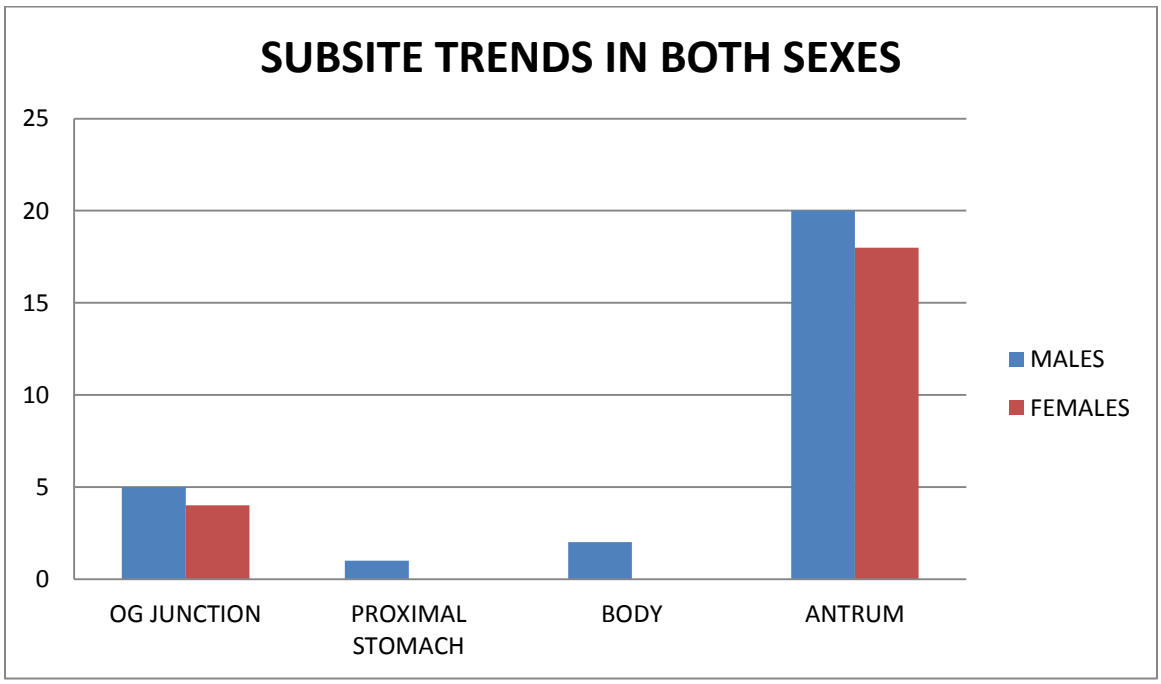


SUBSITES: Table 14; Graph 9

The antrum was the most common site of affliction accounting for 38 (76%) of all subsites. This was also similar in both the sexes with 20 males and 18 females. Oesophagogastric tumours accounted for 9 (18)% of the cases and were similar in both the sexes. None of the females in this study had cancers of the body and proximal stomach.

Table 14- Sub site specific trends in carcinoma stomach

SUBSITES	TOTAL		MALES		FEMALES	
	CASES	%	CASES	%	CASES	%
OG JUNCTION	9	18	5	17.8	4	18.1
PROXIMAL STOMACH	1	2	1	3.5	0	0
BODY	2	4	2	7.1	0	0
ANTRUM	38	76	20	71.4	18	81.8

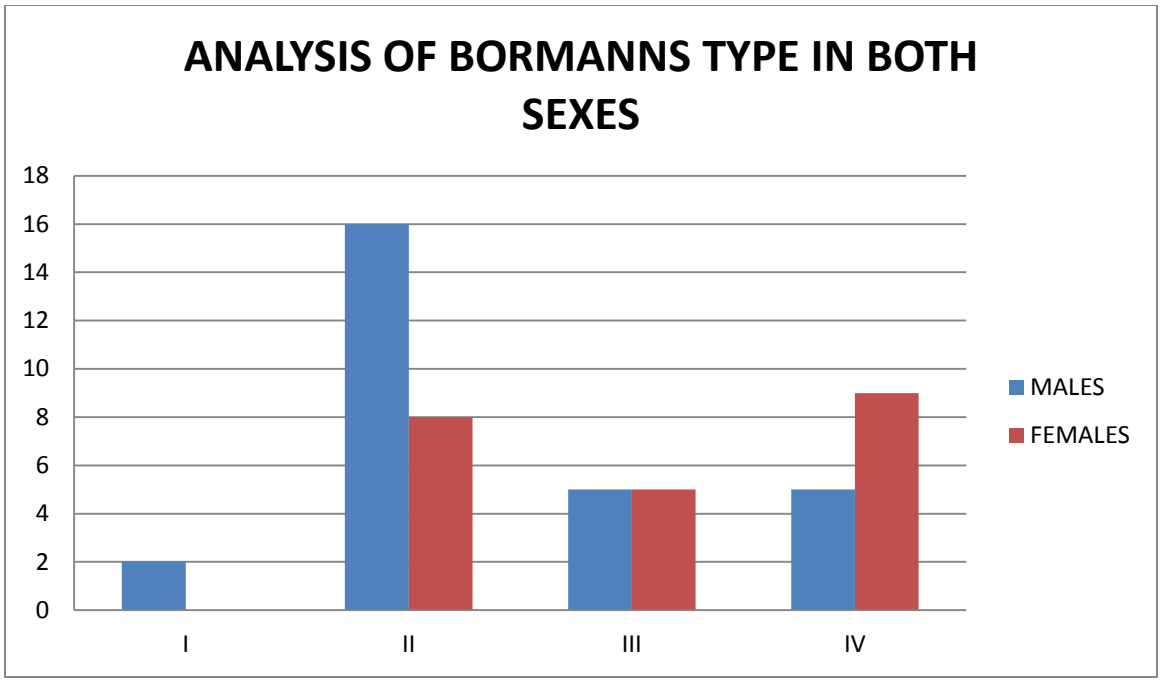


MACROSCOPY: Table 15

The predominant macroscopic subtype was Borrmann type II with 24 (48%) followed by types III and IV. In males the predominant type was Borrmann type II where as in females it was type IV. Females had a higher percentage of locally advanced lesions. There were no Bormann type I lesion in females in this study.

Table 15- Comparison of the macroscopic type in both sexes

BORMANN TYPE	TOTAL		MALES		FEMALES	
	CASES	%	CASES	%	CASES	%
I	2	4	2	7.1	0	0
II	24	48	16	57.1	8	36.3
III	10	20	5	17.8	5	22.7
IV	14	28	5	17.8	9	40.9

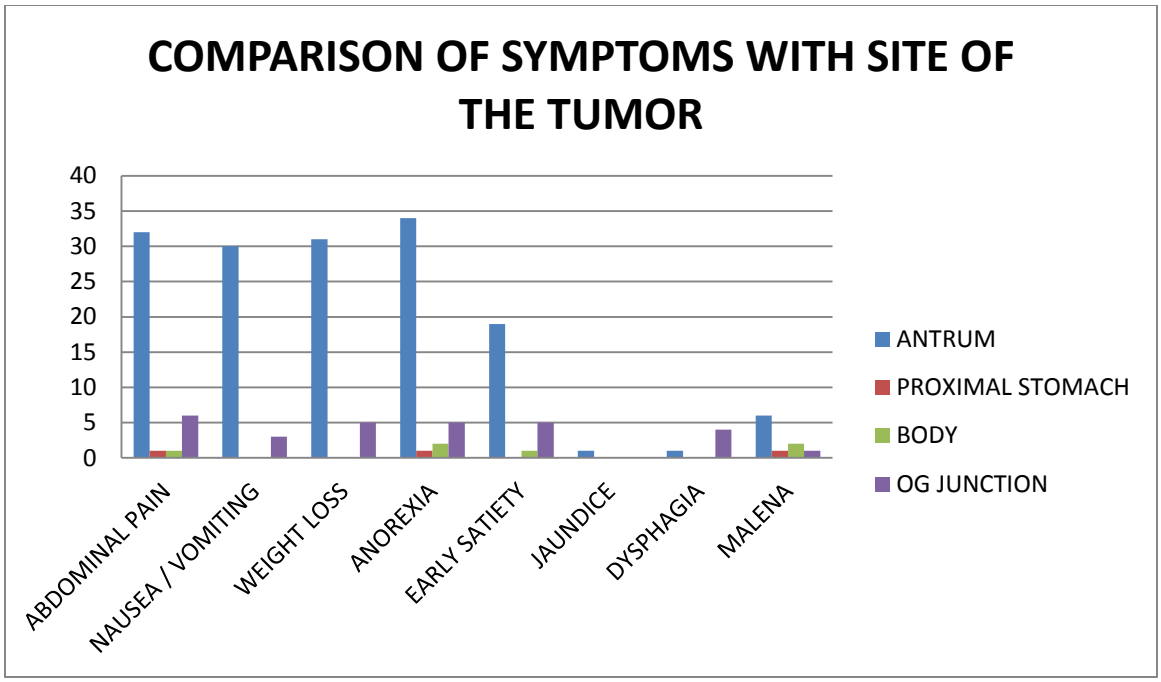


SITE AND SYMPTOMS: Table 16; Graph 10

Antral lesions presented predominantly with nausea/vomiting, weight loss, anorexia and pain abdomen. 50% of the antral growth patients also reported early satiety.

Oesophagogastric tumours had dysphagia as the predominant symptom along with weight loss and anorexia reflecting the aggressive nature of such tumours. Similarly these entire lesions also had dysphagia as a symptom. Malena was more common in lesion of the body followed by the antrum.

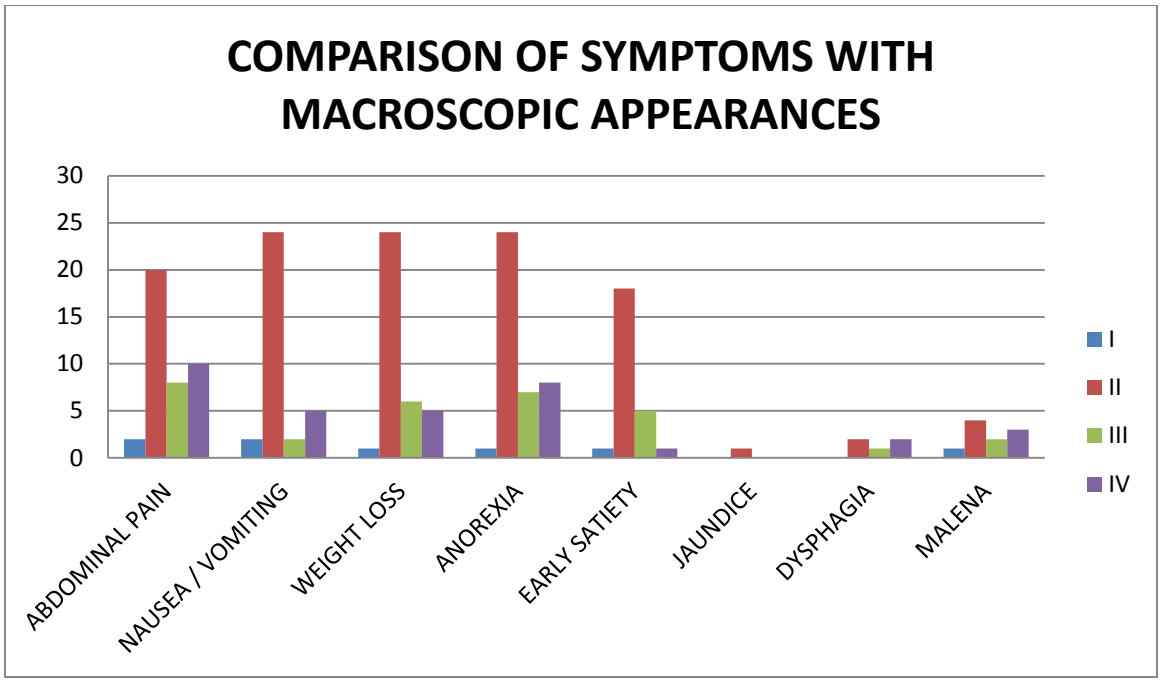
SYMPTOMS	TOTAL CASES	SITE OF TUMOR							
		ANTRUM (38)		PROXIMAL STOMACH (1)		BODY (2)		OG JUNCTION (9)	
		CASES	%	CASES	%	CASES	%	CASES	%
ABDOMINAL PAIN	40	32	84.2	1	100	1	50	6	66.6
NAUSEA / VOMITING	33	30	78.9	0	0	0	0	3	33.3
WEIGHT LOSS	36	31	81.5	0	0	0	0	5	55.5
ANOREXIA	42	34	89.4	1	100	2	100	5	55.5
EARLY SATIETY	25	19	50	0	0	1	50	5	55.5
JAUNDICE	1	1	2.6	0	0	0	0	0	0
DYSPHAGIA	5	1	2.6	0	0	0	0	4	44.4
MALENA	10	6	15.7	1	100	2	100	1	11.1



MACROSCOPY AND SYMPTOMS: Table 17; Graph 11

Borrmann type I lesion patients experienced pain abdomen and vomiting in 100% of cases. All Borrmann type II lesion patients had symptoms of nausea/vomiting, weight loss and anorexia. Type II and III lesion patients had early satiety in 75% and 50% respectively.

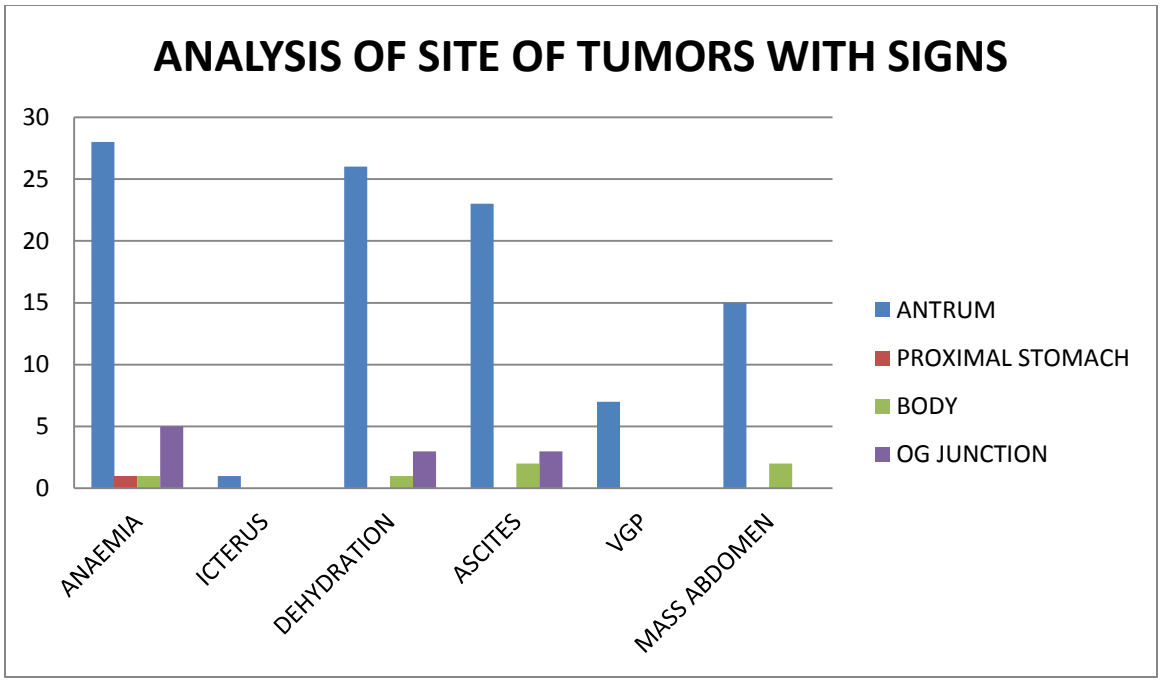
SYMPTOMS	TOTAL CASES	BORMANN TYPES							
		I (2)		II (24)		III (10)		IV (14)	
		CASES	%	CASES	%	CASES	%	CASES	%
ABDOMINAL PAIN	40	2	100	20	83.3	8	80	10	71.4
NAUSEA / VOMITING	33	2	100	24	100	2	20	5	35.7
WEIGHT LOSS	36	1	50	24	100	6	60	5	35.7
ANOREXIA	42	1	50	24	100	7	70	8	57.1
EARLY SATIETY	25	1	50	18	75	5	50	1	7.1
JAUNDICE	1	0	0	1	4.1	0	0	0	0
DYSPHAGIA	5	0	0	2	8.3	1	10	2	14.2
MALENA	10	1	50	4	16.6	2	20	3	21.4



SITE AND SIGNS: Table 18, Graph 12

All cases of proximal tumours had anaemia at presentation. Tumours of the body had ascites at the time of presentation. All the cases with visible gastric peristalsis were antral growths, however only 18.4% of antral growths had visible peristalsis.

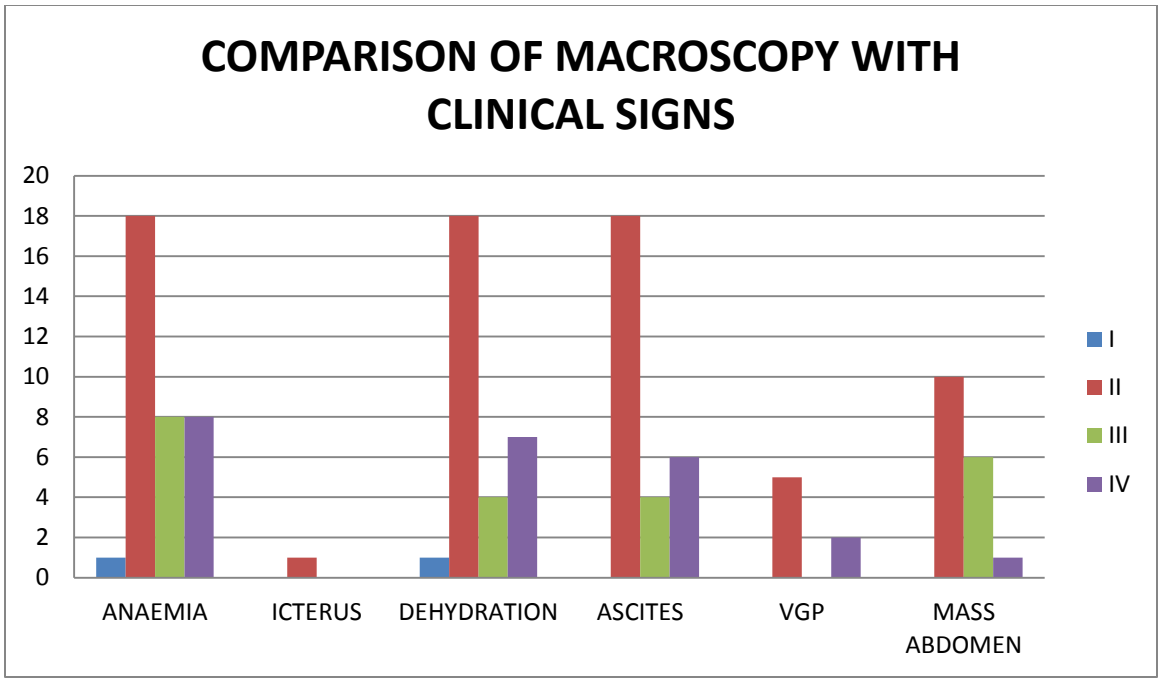
SIGNS	TOTAL CASES	SITE OF TUMOR							
		ANTRUM (38)		PROXIMAL STOMACH (1)		BODY (2)		OG JUNCTION (9)	
		CASES	%	CASES	%	CASES	%	CASES	%
ANAEMIA	35	28	73.6	1	100	1	50	5	55.5
ICTERUS	1	1	2.6	0	0	0	0	0	0
DEHYDRATION	30	26	68.4	0	0	1	50	3	33.3
ASCITES	28	23	60.5	0	0	2	100	3	33.3
VGP	7	7	18.4	0	0	0	0	0	0
MASS ABDOMEN	17	15	39.4	0	0	2	100	0	0



MACROSCOPY AND SIGNS: Table 19; Graph 13

Majority of type II and type III lesions presented with anemia. Borrmann type II, III and IV lesions were more often associated with ascites than type I lesions. Type II and Type III lesions accounted for the majority of cases of mass abdomen.

SIGNS	TOTAL CASES	BORMANN TYPES							
		I (2)		II (24)		III (10)		IV (14)	
		CASES	%	CASES	%	CASES	%	CASES	%
ANAEMIA	35	1	50	18	75	8	80	8	57.1
ICTERUS	1	0	0	1	4.1	0	0	0	0
DEHYDRATION	30	1	50	18	75	4	40	7	50
ASCITES	28	0	0	18	75	4	40	6	42.8
VGP	7	0	0	5	20.8	0	0	2	14.2
MASS ABDOMEN	17	0	0	10	41.6	6	60	1	7.1



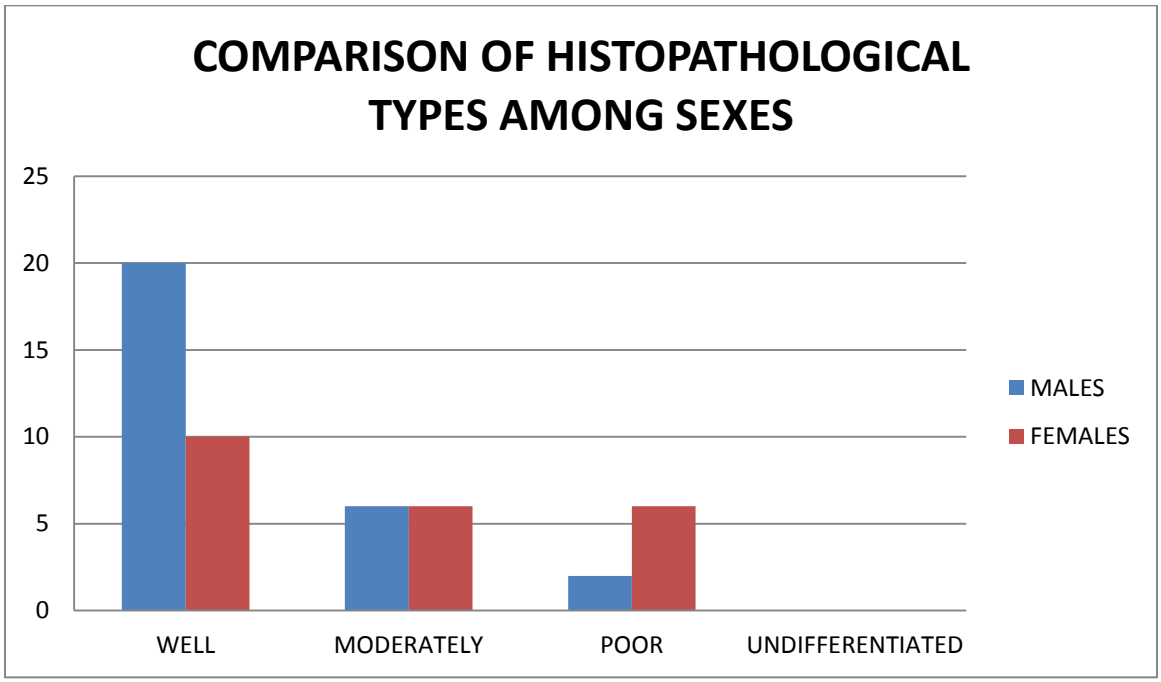
HISTOPATHOLOGY: Table 20, Table 21

Majority of the cases were well differentiated adenocarcinoma. Females had a higher percentage of poorly differentiated tumours.

Table 20- Comparison of histopathology

HISTOLOGY	PRESENT STUDY					
DIFFERENTIATION	TOTAL CASES	%	MALES	%	FEMALES	%
WELL	30	60	20	71.4	10	45.4
MODERATELY	12	24	6	21.4	6	27.2
POOR	8	16	2	7.1	6	27.2

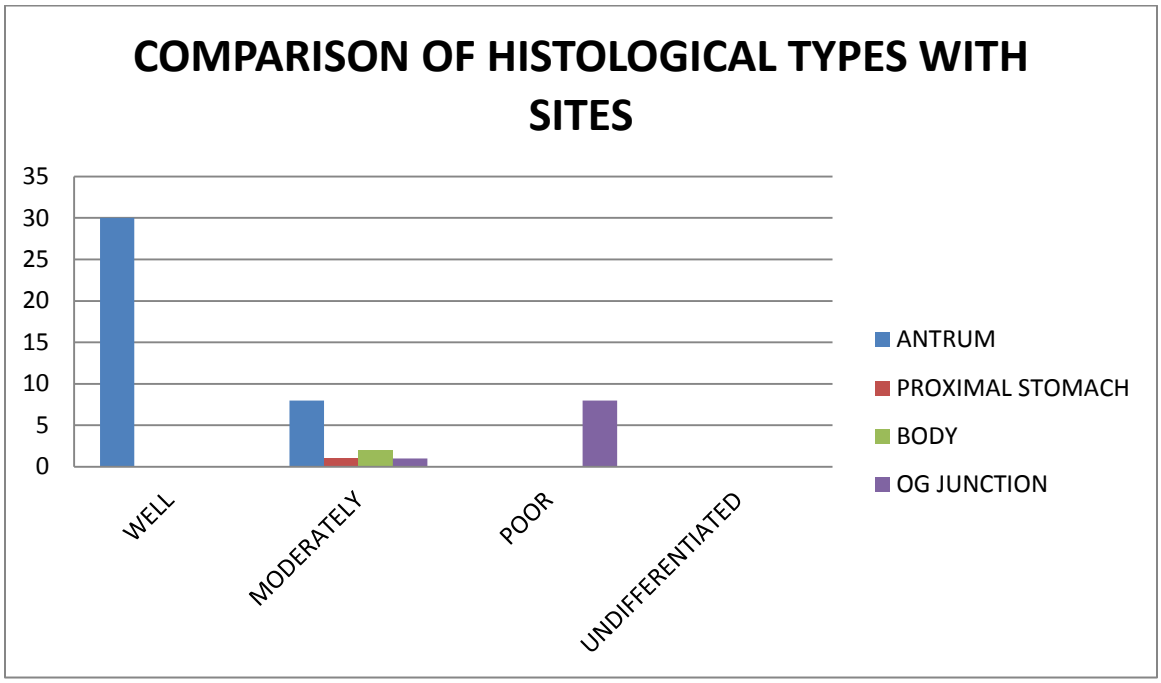
UNDIFFERENTIATED	0	0	0	0	0	0



Antral tumours were predominantly well differentiated and oesophageal had high percentage of poorly differentiated tumours.

Table 21- Comparision of histology with the site of the tumour

HISTOLOGY	PRESENT STUDY				
DIFFERENTIATION	TOTAL	ANTRUM (38)	BODY (1)	PROXIMAL (2)	OG JUNCTION (9)
WELL	30	30	0	0	0
MODERATELY	12	8	1	2	1
POOR	8	0	0	0	8
UNDIFFERENTIATED	0	0	0	0	0



DISCUSSION

This study was undertaken to study the prevalence of gastric cancer as occurring in Government Rajaji Hospital, Madurai which is a tertiary care centre with a large input of cases from Madurai and its surrounding districts.

The study had certain drawbacks. The association of H. Pylori with gastric carcinoma was not studied. Since histopathological confirmation was an inclusion criteria for the study, many suspected cases were not included for lack of definite tissue diagnosis.

Although CT abdomen is recommended for the staging of the disease, it was not performed in most of the cases due to financial constraints. Many cases were referred to the regional cancer institute for further treatment.

Observations were made in this study with M:F ratio of 3:1. These observations are comparable to similar studies in India.

Table 21. Sex distribution among carcinoma stomach patients

SEX	GAJALAKSHMI ET AL 1995		SUMATHI ET AL 2009		PRESENT STUDY	
	CASES	%	CASES	%	CASES	%
MALE	287	73.9	64	71.9	28	56
FEMALE	101	26.1	25	28.1	22	44

In this study maximum no of cases was seen after the age of 45 years. In this study the youngest patient was aged 29 and the oldest 78.

Table 22. Age distribution among carcinoma stomach patients

AGE	GAJALAKSHMI C K ET AL 1996		SEN ET AL 2002		PRESENT STUDY	
	CASES	%	CASES	%	CASES	%
<30	2	0.5	0	0.1	1	2
30-39	30	7.7	7	0.5	7	14
40-49	64	16.5	1	2.2	9	18
50-59	84	21.6	20	7.2	15	30

60-69	124	32	53	19.2	12	24
70-79	68	17.5	77	28.0	6	12
80+	16	4.1	120	43.4	0	0

As the study was conducted in a Government Hospital majority of the cases belonged to the low socioeconomic status accounting for 75% of the cases. The scenario is similar across India where majority of the population belong to the low socioeconomic group further contributing evidence of dietary role of carcinogens. Studies at Chennai and other parts of the country have shown consistent correlation between the lower socioeconomic group and higher prevalence of gastric cancer.

Table 23- Socioeconomic groups among gastric cancer patients

SOCIOECONOMIC STATUS	GAJALAKSHMI ET AL 1995		SUMATHI ET AL 2009		PRESENT STUDY	
	CASES	%	CASES	%	CASES	%
LOW	301	77.5	70	78.6	40	80
MIDDLE	87	22.5	19	21.4	10	20
HIGH	0	0	0	0	0	0

The association of blood group A is well known and the findings were compared with other studies.

STUDY	BLOOD GROUP			
	A (%)	B (%)	O (%)	AB (%)
KAMLESH GULERIA ET AL, PUNJAB	1 (12.5)	5 (62.5)	2 (25)	0 (0)
JOSE ET AL, KERALA	26 (37.1)	14 (20)	23 (32.85)	7 (10)
PRESENT STUDY	23 (46)	9 (18)	12 (24)	6 (12)

Gastric cancer is known to be associated with several environmental risk factors of which diet has an important role. The association of diet has been studied in many studies and consistent results obtained all over. The findings of this study were compared with Sumathi et al. Majority of the patients were non vegetarians in both the studies.

The association of tobacco use and alcohol has been studied. In this study 22(44%) of the patients reported to the use of smoking for a significant periodic compared to 40.5 patients in study by Sumathi et al. Betelnut chewing seen more in females was seen in 16% of the patients compared to 10.2% in the other study.

Table 25- Comparision of risk factors between males and females

FACTORS	SUMATHI ET AL		PRESENT STUDY	
	CASES	%	CASES	%
MIXED DIET	89	100	45	90
VEGETARIAN DIET	0	0	5	10
GREEN LEAFY VEG	58	65.2	40	80
FRUITS	39	43.8	40	80
HIGH SALT INTAKE	0	0	32	64
SMOKED	0	0	15	30

FOODS				
SPICY FOODS	0	0	40	80
SMOKING	36	40.5	22	44
ALCOHOL	32	35.9	22	44
BETEL NUT	9	10.2	8	16

Abdominal pain was major symptom reported in 40(80%) of the cases compared to 56.6% in a study by Safaee et al. Weight loss was seen in 36(72%) of cases compared to 57.7 cases. These findings suggest that patients in our set up present in an early stage of the disease with both local and regional spread. The percentage of patients presenting with malaena was comparable in both groups.

Table 26 Symptom analysis in patients of carcinoma stomach

SYMPTOMS	SAFEE ET AL 2009		PRESENT STUDY	
	CASES	%	CASES	%
ABDOMINAL PAIN	425	56.6	40	80
NAUSEA & VOMITING	324	43.2	33	66
WEIGHT LOSS	434	57.7	36	72
ANOREXIA	-	-	42	84
EARLY SATIETY	263	31.5	25	50
JAUNDICE	-	-	1	2
DYSPHAGIA	263	31.5	5	10
MALENA	144	19.1	10	20

The west has noted a paradigm shift in site of gastric cancer tumours with a steady increase in tumours of the cardium and proximal tumours and a decline in distal tumours. In this study distal tumours continued to be the most common site of affliction with 38(76%) cases and only 9 cases(18%) of proximal tumours. Cherian

et al 8 studying a 16 year trend of gastric cancer at Chennai also had similar findings.

Table 27 Sub site specific trends in carcinoma stomach

SUB SITE	CHERIAN ET AL		PRESENT STUDY	
	CASES	%	CASES	%
OG JUNCTION	65	3.78	9	18
PROXIMAL STOMACH	97	5.64	1	2
BODY	400	23.27	2	4
ANTRUM	1157	67.31	38	76

In this study majority of the tumours were well differentiated. Moderately and poorly differentiated were equally differentiated. In the study by Safee et al poorly differentiated tumours were more common.

Table 28 Comparision of histology according to Broder's classification

HISTOLOGY	SAFEE ET AL		PRESENT STUDY	
DIFFERENTIATION	CASES	%	CASES	%
WELL	113	23	30	60
MODERATELY	142	30.1	12	24
POOR	203	43	8	16
UNDIFFERENTIATED	14	3	0	0

CONCLUSION

This study was undertaken to study the prevalence of gastric cancer as occurring in Government Rajaji Hospital, Madurai which is a tertiary care centre with a large input of cases from Madurai and its surrounding districts.

In this study gastric cancer was more prevalent in males.

Majority of the patients belonged to the lower socioeconomic strata and had association of risk factors. Blood group A was the prevalent blood group.

The disease was more prevalent in patients above the age of 45 with the oldest being 78. Majority presented in the advanced stage of the disease although there were few cases which presented in the early gastric carcinoma stage.

The pylorus remained the most common site of affliction in contrast to western countries which have showed a consistent shift towards proximal tumours. The major percentage of the tumours was well differentiated.

The findings of this study are comparable to other similar studies in India and

proximal gastric tumours continue to be the major subtype in this part of the world and association of risk factors increase the likelihood of an individual developing gastric cancer.

SUMMARY

Gastric cancer is a disease of the older age group.

The association of risk factors is well known and consistent and hint at the primordial prevention of the disease.

Distal tumours continue to be the major subtype in this study. Successful preventive strategies have to be developed a multischolastic approach should combine population screening with molecular biological techniques that are being developed.

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ANNEXURES

"CLINICAL STUDY ON INCIDENCE,PATHOLOGICAL PATTERN AND MANAGEMENT OF GASTRIC CARCINOMA IN GOVT. RAJAJI HOSPITAL,MADURAI

PROFORMA

NAME CASE NO:

AGE D.O.A

SEX D.O.S

OCCUPATION D.O.D

ADDRESS HOSPITAL

SOCIOECONOMIC STATUS

CHIEF COMPLAINTS:

Mass per abdomen:

Pain abdomen:

Distension:

Loss of weight:

Loss of apetite:

Localized swelling:

Fever:

Jaundice:

Others:

HISTORY OF PRESENT ILLNESS:

1) MASS PER ABDOMEN

Onset:

Site:

Duration:

Progression- sudden increase/sudden decrease/gradual increase/no progression:

Secondary changes:

Similar swellings elsewhere

2)PAIN ABDOMEN:

Onset:

Duration:

Nature-dragging/colicky/dull aching/continuous

Intensity:mild/severe

Progression/nonprogression

Site:localized/generalized

Aggravating/relieving factor

Relation to food

Vomiting

Radiation

3)DISTENSION:

Onset:

Duration:

Progression:

Relation to vomiting

Relation to pain

4)JAUNDICE

5)CHEST PAIN

Site

Duration

Nature

6)COUGH/HEMOPTYSIS

7)FEVER

8)LOSS OF WEIGHT:

9)OTHERS:

PAST HISTORY

Any sImilar complaints

Any previous treatment/surgeries

History of TB / DM / HTN / COPD

PERSONAL HISTORY

Apetite	sleep
Diet	bowel and bladder habits
Smoker	alcoholic

FAMILY HISTORY

MENSTRUAL HISTORY & OBSTETRIC HISTORY(females)

DRUG HISTORY:

GENERAL PHYSICAL EXAMINATION:

Apperence	vital signs
Pallor	pulse
Cyanosis	blood pressure
Clubbing	respiratory rate
Jaundice	temperature
Pedal edema	
Lymphadenopathy	

SYSTEMIC EXAMINATION:

EXAMINATION OF ABDOMEN:

A) INSPECTION:

Contour of abdomen- fullness/mass-site

Flat

Umbilicus

- position: stretched/everted/pushed

Movements with respiration:

Visible pulsations/peristalsis

Dilated veins

Flanks

Skin

Hernia orifices

External genitalia

Renal angles

Supraclavicular fossa

any mass:

- number
- shape

size

site

extent

surface

borders

impulse on coughing

presseure effects

pulsations

B)PALPATION:

Local temperature

Tenderness:site

Mass

Number

Shape

Size

Site

Extent

Surface

Borders

Movement with respiration

Consistency : soft / cystic / firm / hard

Pulsations

Mobility: free/restricted

Vertical/horizontal

Plane: parietal / intraperitoneal / retroperitoniel

Organomegaly

Guarding/rigidity

Palpation of hernial orifices

External genitalia

Any other mass

c) PERCUSSION

percussion note over mass

whole abdomen:reonant/dull

free fliud:normal /obliterated

dullness over renal angle

D)AUSCULTATION:

Bowel sounds

Bruit

E) PER RECTAL

F) PER VAGINAL

G) EXTERNAL GENITALIA

H) SPINAL EXTREMITIES

I) SUPRACLAVICULAR LYMPHNODES

RESPIATORY SYSTEM

CARDOVASCULAR SYSTEM

CENTRAL NERVOUS SYSTEM

MUSCULOSKELETAL SYSTEM

CLINICAL DIAGNOSIS:

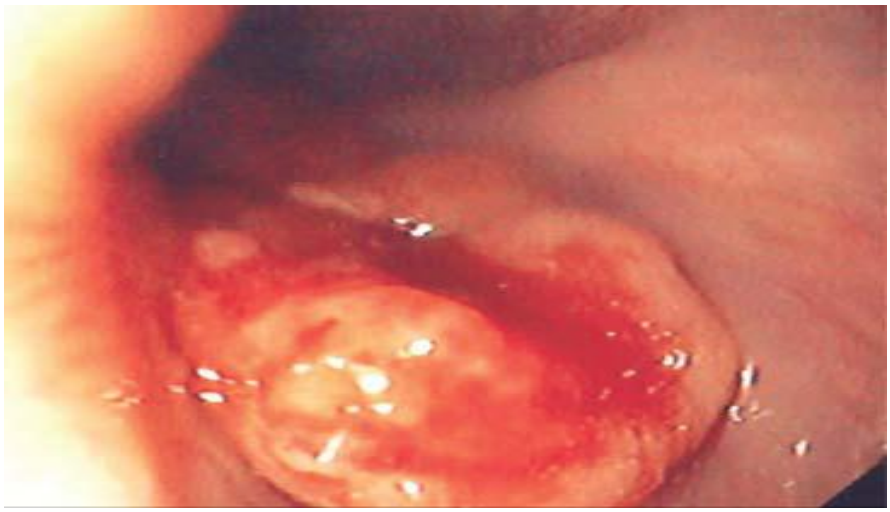

Fig. 7 Endoscopic photograph showing ulceroproliferative growth in antrum

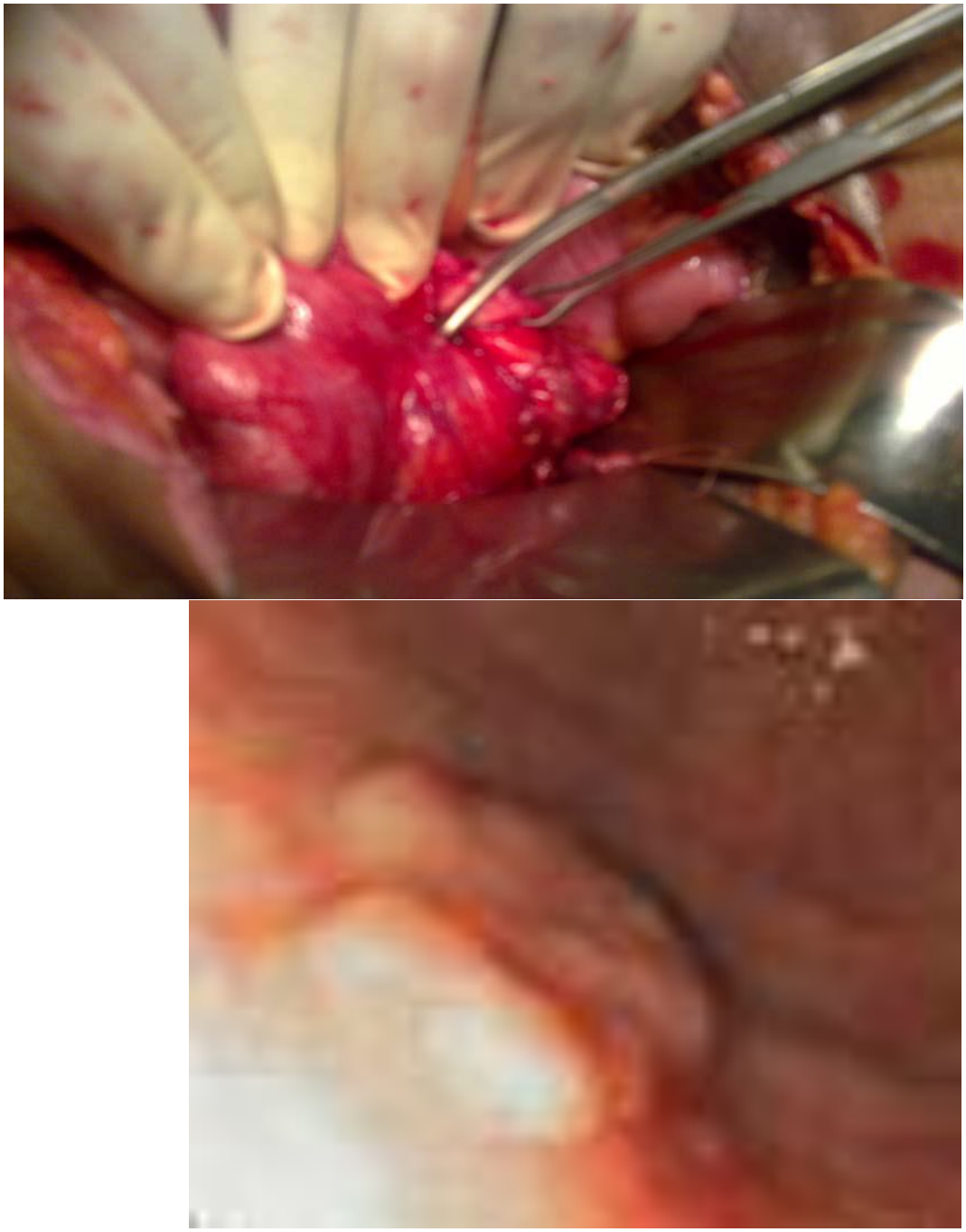

Fig.8 Endoscopic photograph showing early gastric cancer

Fig. 9 Photograph showing gastric cancer invading the posterior wall and perforating it

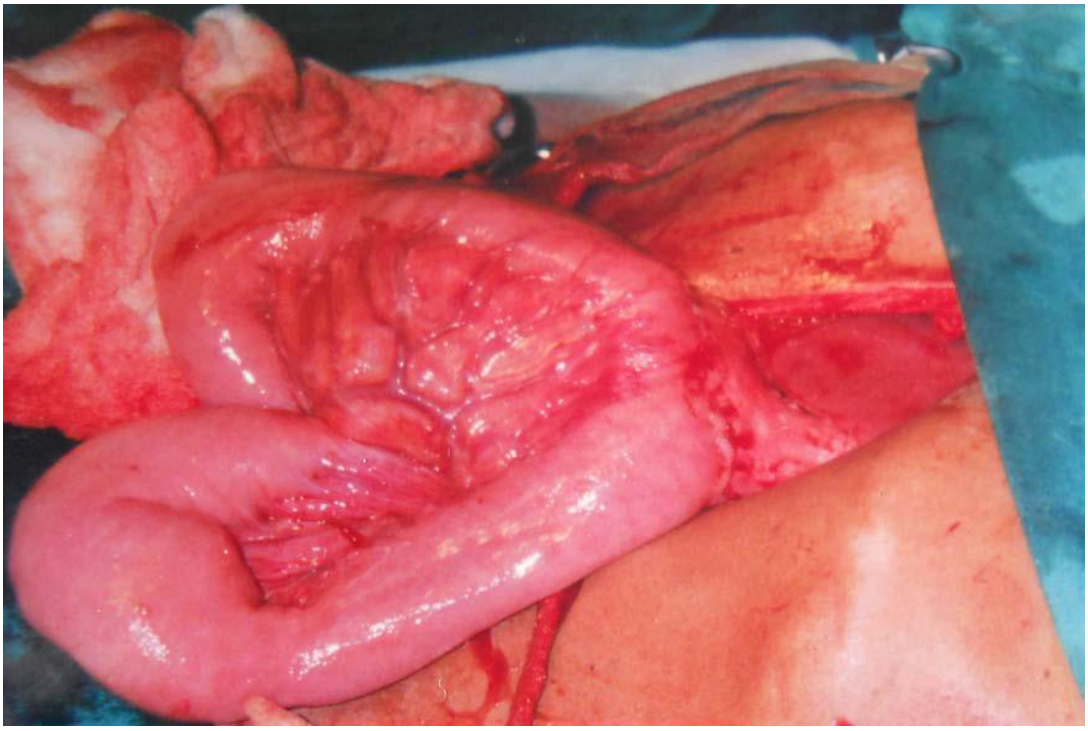

Fig. 10 Photograph showing palliative anterior gastrojejunostomy 99

Fig. 11 Photograph showing partial gastrectomy specimen

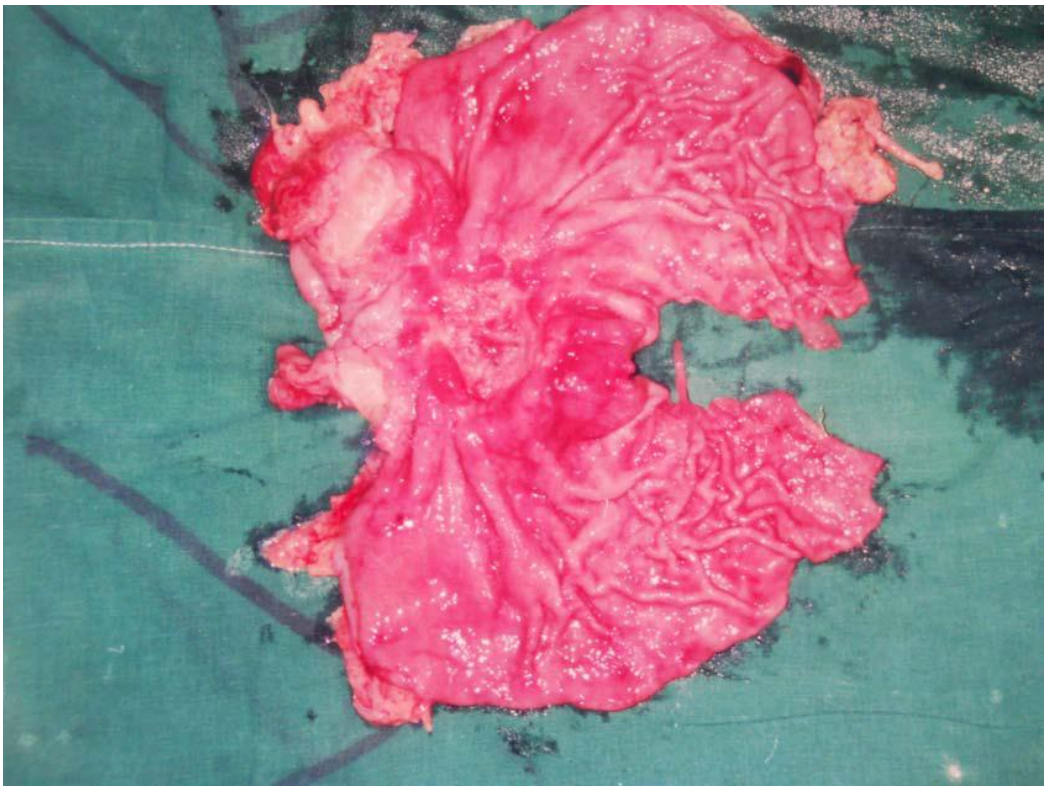

Fig12 Photograph showing opened partial gastrectomy specimen100

NO.	NAME	AGE	SEX	IP.NO	PLACE	DOA	DOS	DOD	SYMPTOMS	SE STATUS	DIET	SMOKED F	SPICY	FRUITS
1	MARIAMMAL	50	F	11579	MADURAI	2.8.13	4.8.13	12.8.13	abcde	LOW	MIXED	-	+	+
2	LINGAM	70	M	82977	VIRUDHUNAGAR	5.8.13	7.8.13	15.8.13	abcde	LOW	MIXED	-	+	+
3	MUNIYANDI	65	M	2777	ARUPPUKOTTAI	17.8.13	19.8.13	27.8.13	bdeh	LOW	MIXED	+	+	+
4	MUTHUKARUPPAN	58	M	14612	DINDUGAL	21.8.13	23.8.13	1.9.13	abdch	MID	MIXED	-	-	-
5	KALUVAYEE	58	F	21245	KOTTAMPATTI	29.8.13	31.8.13	9.9.13	acd	LOW	MIXED	+	+	+
6	SANJEEVI	54	M	21234	MADURAI	7.9.13	9.9.13	17.9.13	abcd	MID	MIXED	-	+	+
7	LAKSHMI	42	F	26999	KOTTAMPATTI	10.9.13	12.9.13	20.9.13	adeg	LOW	MIXED	-	+	+
8	VELUTHAAI	45	F	31945	DINDUGAL	17.9.13	19.9.13	27.9.13	abc	LOW	MIXED	-	+	+
9	MEYYAPPAN	60	M	33972	ARUPPUKOTTAI	24.9.13	26.9.13	4.10.13	acde	LOW	MIXED	+	+	-
10	ARUMUGAM	59	M	49199	VIRUDHUNAGAR	5.10.13	7.10.13	15.10.13	abcdg	MID	MIXED	-	-	+
11	MOHAN	62	M	50947	SIVAKASI	9.10.13	11.10.13	19.10.13	beh	LOW	MIXED	+	+	-
12	MANICKAM	50	M	50128	ARUPPUKOTTAI	19.10.13	21.10.13	29.10.13	acdh	LOW	MIXED	-	-	+
13	PAPPA	52	M	76215	MADURAI	22.10.13	24.10.13	2.11.13	bcd	LOW	MIXED	-	+	+
14	KAMATCHI	60	F	83629	SIVAGANGAI	2.11.13	4.11.13	12.11.13	aceh	MID	MIXED	+	-	+
15	RAMAN	55	M	723	DINDUGAL	7.11.13	9.11.13	17.11.13	abd	LOW	MIXED	-	+	-
16	PALANISAMY	66	M	93863	SIVAGANGAI	9.11.13	11.11.13	19.11.13	abe	LOW	VEG	-	+	+
17	KALEESWARI	35	F	1860	KOTTAMPATTI	15.11.13	17.11.13	25.11.13	bcd	LOW	MIXED	-	+	+
18	MARAVARMAN	50	M	9107	VIRUDHUNAGAR	21.11.13	23.11.13	1.12.13	ace	MID	MIXED	+	+	+
19	PEER MOHAMMED	70	M	12045	ARUPPUKOTTAI	24.11.13	26.11.13	4.12.13	abd	MID	MIXED	-	-	-
20	PANDIYARAJA	67	M	13600	SIVAKASI	6.12.13	8.12.13	16.12.13	acd	LOW	MIXED	-	-	+
21	ANNAMALAI	75	M	17451	MADURAI	9.12.13	11.12.13	19.12.13	abcdh	LOW	MIXED	+	+	+
22	JANARTHANAN	56	M	40884	MADURAI	17.12.13	19.12.13	27.12.13	adeh	LOW	MIXED	+	+	+
23	KARUPPAYYA	61	M	44498	KOTTAMPATTI	25.12.13	27.12.13	5.1.14	abcg	LOW	MIXED	-	+	+
24	NATARAJ	65	M	51369	DINDUGAL	2.1.14	4.1.14	12.1.14	abd	LOW	MIXED	-	+	+
25	NAGALAKSHMI	45	F	58329	ARUPPUKOTTAI	6.1.14	8.1.14	16.1.14	abch	MID	MIXED	-	+	-
26	SILAYAPPAN	78	M	70926	SIVAGANGAI	10.1.14	12.1.14	20.1.14	abdgh	LOW	MIXED	+	+	+
27	CHINNAIRULAN	61	M	90521	VIRUDHUNAGAR	14.1.14	16.1.14	24.1.14	bce	LOW	MIXED	-	+	+
28	PANDIYAMMAL	58	F	5224	SIVAKASI	20.1.14	22.1.14	30.1.14	acd	LOW	VEG	-	-	+
29	RASAKILI	56	M	17937	KOTTAMPATTI	22.1.14	24.1.14	2.2.14	abd	MID	MIXED	+	+	-
30	THONDHIPILLAYAR	29	M	32105	MADURAI	29.1.14	31.1.14	9.2.14	abced	LOW	MIXED	-	+	+
31	GOPALAKRISHNAN	38	M	44739	VIRUDHUNAGAR	2.2.14	4.2.14	12.2.14	bc	LOW	MIXED	-	+	+
32	PAPPATHY	52	F	43499	ARUPPUKOTTAI	7.2.14	9.2.14	17.2.14	abde	LOW	MIXED	-	+	-
33	KUMARAYEE	65	F	66121	SIVAGANGAI	17.2.14	19.2.14	27.2.14	ad	LOW	VEG	-	-	+
34	VEMBULAKKAL	38	F	73509	KOTTAMPATTI	26.2.14	28.2.14	6.3.14	acf	LOW	MIXED	-	+	+
35	REVATHY	36	F	69916	MADURAI	1.3.14	3.3.14	11.3.14	abdc	LOW	MIXED	-	+	+
36	ANNAVI	45	F	73570	SIVAGANGAI	6.3.14	8.3.14	16.3.14	ade	LOW	MIXED	+	+	+
37	KAATTURANI	32	F	74160	ARUPPUKOTTAI	11.3.14	13.3.14	21.3.14	abc	LOW	MIXED	-	+	-
38	ANNAKILI	48	F	72454	MADURAI	21.3.14	23.3.14	31.3.14	bcd	LOW	MIXED	-	-	+
39	PONNUTHAAI	60	F	68467	SIVAGANGAI	25.3.14	27.3.14	5.4.14	ace	LOW	MIXED	+	+	+
40	KAALIAMMAL	78	F	62358	VIRUDHUNAGAR	5.4.14	7.4.14	15.4.14	abcd	LOW	MIXED	-	+	+
41	CHELLAMUTHU	45	M	77973	SIVAKASI	13.4.14	15.4.14	23.4.14	cde	LOW	MIXED	-	+	+
42	KANNAMMAL	60	F	77997	DINDUGAL	21.4.14	23.4.14	1.5.14	abh	LOW	MIXED	-	+	+
43	THAVAMANI	42	F	73943	SIVAKASI	4.5.14	6.5.14	14.5.14	acbhd	MID	MIXED	+	+	+
44	GANESAN	49	M	78725	ARUPPUKOTTAI	12.5.14	14.5.14	22.5.14	ace	LOW	VEG	-	+	+
45	POTHUMANI	36	F	78738	VIRUDHUNAGAR	19.5.14	21.5.14	29.5.14	bcd	LOW	MIXED	+	+	-
46	NATARAJAN	59	M	80596	SIVAGANGAI	22.5.14	24.5.14	2.6.14	bde	LOW	MIXED	-	-	+
47	RAAKKU	40	F	3600	DINDUGAL	13.6.14	15.6.14	23.6.14	abcd	LOW	MIXED	-	+	+
48	VELLAISAMY	77	M	83665	SIVAGANGAI	23.6.14	25.6.14	3.7.14	ade	MID	VEG	+	+	+
49	DHANABALAN	50	M	83755	MADURAI	7.7.14	9.7.14	17.7.14	abcdg	LOW	MIXED	-	+	+
50	PUSHPAM	30	F	21995	MADURAI	15.7.14	17.7.14	25.7.14	aeh	LOW	MIXED	-	+	+

KEY TO MASTER CHART

APL- Above Poverty Line
BPL- Below poverty Line
NS- Nothing Significant
M- Mix ed
V- Vegetarian
F- Frequent
O- Occasional
Mo- Moderate
A- Ascites
D- Deh ydration
PA- Pyloric Antrum
PALN- Paraaortic Lymph nodes
PE- Pleural effusion
DS- Dilated Stomach
EL- Emergency Laparotomy
F- Fundus
PO- Pyloric Obstruction
GEJ- Gastro-oesophageal junction
SYMTOMS

a- Abdominal pain
b- Nausea/vomiting
c- Weight loss
d- Anorexia
e- Early satiety
f- Jaundice
g- Dysphagia
h- malena

I- Infiltrative
IO- Inoperable
JJ- Jejunojejunostomy
LC- Lesser curvature
LS- Liver secondaries
MA - Minimal Ascites
MRD- Medical Renal Disease
O- Operable
P- Poor
B- Body
C- Cardia
D- Diffuse
DC- Decreased capacity
Per- Perforation
Po- Polypoidal
FJ- Feeding Jejunostomy
G- Growth
GC- Greater curvature